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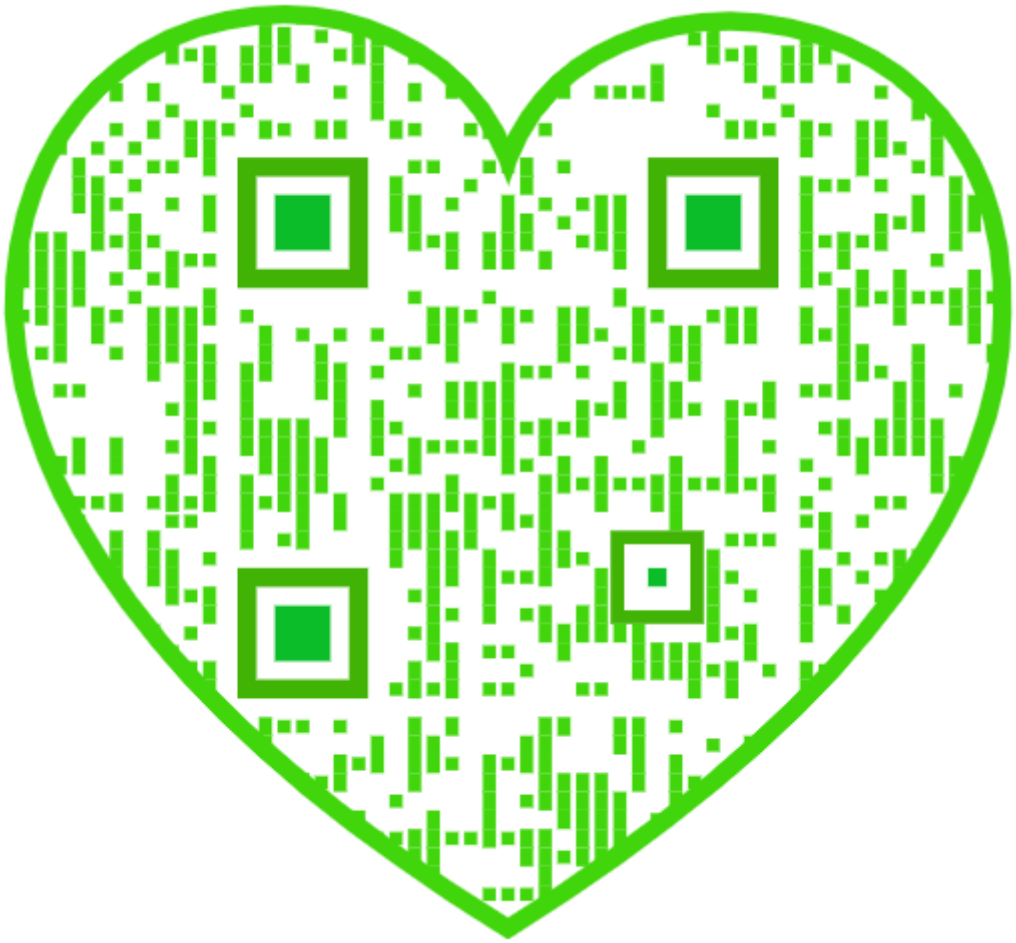

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INTERNAL MEDICINE

A WORK FOR THE PRACTICING PHYSICIAN ON DIAGNOSIS AND TREATMENT WITH A COMPLETE DESK INDEX

BY
NATHANIEL B. POTTER, M.D., JAMES C. WILSON, M.D.

IN THREE VOLUMES

ILLUSTRATED WITH 427 TEXT ILLUSTRATIONS AND 14 IN COLOR

MEDICAL DIAGNOSIS

FIFTH EDITION REVISED AND ENLARGED IN TWO VOLUMES

Vol. I

MEDICAL DIAGNOSIS IN GENERAL THE METHODS AND THEIR IMMEDIATE RESULTS; SYMPTOMS AND SIGNS, TESTS

BY
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ASSISTED BY
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To

THE MEMORY OF MY

FATHER

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PREFACE TO THE FIFTH EDITION

FOUR years have elapsed since the last edition of this work was issued. To present recent advances in diagnostic methods and their practical application, several sections have been rearranged and much new matter added.

The general considerations which determined the original form of the work and the arrangement of its topics and which account in large measure for its prompt and wide acceptance alike among practitioners and students have not been modified. But careful revision has called for important changes. Professor Robinson has contributed a new section on Graphic Methods in the Study of Diseases of the Heart; Professor Sweet has carefully revised and enlarged his section on Diseases of the Eye; Doctor Lloyd has brought the section on Diseases of the Nervous System fully up to date; Doctor Rehfuss has written one upon the newer Gastro-Enterological methods with especial reference to the Fractional Study of the Contents of the Stomach and Duodenum, and Doctor Corson White, a very practical section on Serology.

Focal Infection, Vital Function Testing Methods and their Significance, Acidosis, Dehydration and Allergy, Anaphylaxis and Serum Sickness have been included among the added subjects, together with many important minor matters incorporated in the text. Nosological rearrangements concerning Diseases of the Heart, Diseases due to Vitamins, Disorders of Metabolism and those arising in consequence of Deranged Functions of the Endocrine Glands have been made in response to the requirements of present views. Many new illustrations have been inserted.

The division of the work into two separate volumes made necessary by the foregoing changes and its publication in connection with Ortner's Treatment of Internal Diseases as edited by Professor Potter greatly increase its value alike to the practitioner and the student; while the separate general index is a practical aid to the use of the three volumes.

My thanks are due to Dr. Creighton H. Turner for efficient assistance in seeing the work through the press and to Messrs. J. B. Lippincott Company for untiring and generous coöperation.

J. C. WILSON.

PHILADELPHIA, April, 1919.

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PREFACE TO THE FIRST EDITION

THIS volume has been written partly in response to the wishes of some of my professional colleagues, partly to meet the urgent demands of many successive classes of pupils, but chiefly in the hope that at this time a convenient and practical presentation of the subject of Medical Diagnosis will prove useful to the profession at large.

The treatment of the subject-matter under four main headings has been adopted with the view of simplifying the arrangement of the topics in a department of medicine which has attained large scope and insistent importance. It is the confident expectation of the author that this plan will fulfil the twofold requirement, that, within the compass of a single book, clinical phenomena, on the one hand, and, on the other, those complexes of clinical phenomena which constitute diseases, are brought into correlation in such a manner that the practitioner who seeks information upon an obscure case may at once turn to the discussion of the methods available to clear it up, and the student may find the definite clinical applications of the same methods and their results in descriptive medicine.

Practical rather than theoretical considerations have been held constantly in view alike in the treatment of the clinical and the laboratory subjects. To attain this end a degree of positiveness of assertion not warranted under other circumstances and the avoidance of the discussion of moot and unsettled questions have seemed proper.

The Medical Diagnosis of J. M. Da Costa was published in 1864. That brilliant contribution to the literature marked an epoch in the progress of internal medicine. From the time of its appearance the traditional conception of diagnosis by intuition—a gift of the favored few—ceased to occupy the thoughts of medical men, and the subject ranged itself among the arts based upon scientific facts. It maintained in successive editions during the life of its distinguished author its position in the forefront of the progress of applied medicine during a period of extraordinary advancement in the collateral sciences upon which the practice of medicine rests. The continuing rapid development of knowledge relating to the facts of

medicine in the last decade has rendered necessary fresh presentations of the subject, and from time to time excellent works have appeared. These differ greatly among themselves, according to the views of their several authors, in method and detail. To add to this honorable list demands the justification of something different in method, new arrangement of detail, and the presentation of the whole subject in accordance with the requirements of contemporary medicine. It is hoped that in the present volume these demands are fulfilled. It is the outcome of many years devoted to work in the wards, with the controlling side-lights upon bedside diagnosis afforded by the clinical laboratory, revelations at the hands of surgical colleagues in the operating theatre and confrères in pathology in the post-mortem room, the frequent opportunity of seeing unusual and grave cases in consultation, and long experience as a teacher. Such a career arouses enthusiasm but begets caution. It does not encourage in any way the belief that diagnosis in medicine is an easy matter, but forces the conclusion that it is often difficult and in rare instances impossible. For this reason and because we are always eager to extend the boundaries of our knowledge, this art is as absorbing as it is useful.

In the making of a handbook of this kind it is necessary to draw at every step upon the great fund of acquired information which has become the common property of the profession. To those whose contributions have formed that fund and to those who are daily adding to it I tender grateful acknowledgment for its use. I have mentioned by name those to whose work I have especially referred, but, as a general rule, it has been impracticable for want of space to append systematic references to the literature.

The illustrations are in large part drawn from personal observations. They have been selected solely with the view to elucidate the subject in hand. Diagrams have been employed when this method of presentation has appeared desirable, and the free use of clinical charts constitutes an important feature of the work.

To the friends and fellow-workers who have rendered—some small, some larger, but all generous and willing—assistance, I desire to express my thanks. The list includes many colleagues in the hospitals with which I am connected, some who were and others who still are resident physicians. It includes also Mr. Wilbert and Drs. Bachmann, Manges, Rosen-

berger, Rowntree, Royer, White, W. R. Wilson, and J. Leslie Davis. To Drs. de Schweinitz, Welch and Schamberg, T. M. Rotch, Packard, Piersol, Young, Emerson, Dudley Fulton, and many others, together with their publishers, I am indebted for permission to use illustrations. The pages on the diagnosis of diseases of the eye were written by Dr. Sweet; those on the stomach and intestines mainly by Dr. Gwyn; those on the nervous system by Drs. James Hendrie Lloyd and the late William Pickett; those on X-ray diagnosis by Dr. Moore, and those on the examination of the blood, urine, sputum, and other fluids by Dr. F. J. Kalteyer. The excellent drawings, plates, and other illustrations made by Messrs. Schmidt and Faber add much to the usefulness of the book.

I am under special and lasting obligation to Dr. Kalteyer for his most able and untiring aid while the work was in press, and to the publishers for their generous coöperation at every stage in its making.

J. C. Wilson.

Philadelphia, September, 1909.

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A HANDBOOK
of
MEDICAL DIAGNOSIS

VOLUME I

DIAGNOSIS IN GENERAL; THE METHODS; SYMPTOMS AND SIGNS; TESTS.

PART I.

OF MEDICAL DIAGNOSIS IN GENERAL.

I.

GENERAL CONSIDERATIONS.

Diagnosis in medicine is the art or process of distinguishing between different diseases. It occupies a position related on the one hand to etiology—that science which has for its object the study of the causes of disease—and on the other to therapeutics—the art of healing. To recognize a disease involves the consideration of its causes, and if they can be corrected or removed, points the way to a cure—*causa sublata tollitur effectus*. Even when the causes are beyond our control or the lesions which they have produced are permanent, a knowledge of the true nature of the malady may enable us to select judiciously the therapeutic measures by which are brought about those adjustments which relieve suffering and prolong life. The maxim, "*qui bene diagnoscit bene curat,*" is not without truth. Finally, a correct diagnosis is essential to a reasonable prognosis, since by this means only can we foretell the probable course of a disease, whether it tends to recovery, to continuing disability, or to death.

Diagnosis is of fundamental importance in scientific medicine. The prevention of disease and the healing of the sick constitute the goal of medicine, but diagnosis is the course by which that goal is to be reached. Empirical systems ignore alike the causal and the pathological basis of disease and content themselves with the study and treatment of symptoms, and all practice tends to degenerate into charlatanism in proportion as it allows itself to be betrayed into this delusion. Rational medicine, on the other hand, regards symptoms primarily as clues to a diagnosis, only secondarily as indications for treatment; and treatment itself as efficient

when it is causal or radical, and as a makeshift when it is simply palliative or symptomatic. When pain is present we seek by the methods of diagnosis to find the cause of it and to relieve it by the removal of the cause, and are not content simply to relieve the pain without regard to the underlying condition which produced it.

The art of diagnosis is important not only because of its practical utility, but also because it deals with the facts of nature. Hypotheses and theories in regard to disease come and go, nosological arrangements change and shift like the colors in the kaleidoscope, therapeutic fashions rise and fall, but the facts gained by close and constant observation belong to science and are changeless, and these are the facts with which diagnosis is concerned. It has been said that the whole art of medicine is in observation. It is certainly true that the art of diagnosis is in observation. Errors occur far more commonly from incomplete observation than from want of knowledge. A systematic, patient, painstaking study of the facts is essential to success.

The requirements of this branch of medicine are most varied and exacting. A knowledge of anatomy, and especially of visceral and regional anatomy, is essential. The variations in the size and position of the organs within the limits of health must be known. The structure and relation of the parts entering into the formation of the nervous system must be mastered. The physiological functions of the complex human organism are to be familiar knowledge. The causes of disease, both those belonging to the outside world and those developed within the body itself, and the susceptibilities which vary at different periods of life and under different circumstances, must be thoroughly understood. Changes produced by pathogenic factors must be clearly known. In truth, the facts of pathology and semeiology and the natural history of the diseases constitute the basis of diagnosis.

Hence, in the arrangement of medical studies, diagnosis is properly taken up after the student has made advanced progress in the fundamental branches, and the success of the practitioner in this field of medicine is dependent upon close habits of observation, accurate knowledge, and large experience. A judicial temperament and the ability to weigh evidence and assign due relative value to the factors in clinical problems are essential. Not less important are patience and a systematic procedure in all cases. Equally essential are correct habits of reasoning, since without these a faulty conclusion may follow accurately observed facts. The diagnostician in the broad field of clinical medicine must frequently turn for assistance to his professional colleague, who is familiar with the facts of the more restricted specialties and has mastered their technic, and he is becoming with advancing knowledge more and more dependent for accurate results upon instruments of precision and the clinical laboratory. Finally, the diagnostician should not be without imagination. Making use of his knowledge of anatomy and morbid anatomy he should cultivate the habit of picturing to himself the changes in the organs of the body by which clinical phenomena are brought about, such as the consolidated lung in pneumonia, the fibrinous exudate or effusion in pleurisy, the impacted gall-stone in biliary fever, the thrombus in phle-

bitis, the clot and its location in cerebral apoplexy; and in order that this habit of forming at the bedside, by a process of projective imagination, mental pictures of structural conditions hidden from the eye may be developed to the greatest extent, he should avail himself of every opportunity of witnessing operations involving the cranium, thorax, and abdomen, and of being present at post-mortem examinations.

The object of diagnosis is not merely to find a name for a morbid condition or symptom-complex. This it does, it is true, but in doing so it determines the condition of the patient as an individual, the intensity of the pathological process, the importance of prominent symptoms, the presence or absence of complications or intercurrent diseases, and in acute maladies the ability of the organism to withstand the attack. A correct diagnosis enables us to determine whether the condition of the patient is due to causes still operative or the result of influences that have ceased to act; whether or not his malady is self-limited, and, by collating the facts of any given case with the general knowledge of the profession, to form an opinion as to the probable duration of the sickness and its ultimate outcome. It informs us whether the prominent symptoms are the direct manifestation of an independent morbid process, as in gonorrhœal arthritis, the expression of a constitutional susceptibility, as in rheumatic fever, or an acute outbreak of a persistent condition, as in podagra. It enables us to recognize primary and secondary morbid processes and to distinguish between them, as in appendicitis and peritonitis, and to perceive the relation between associated visceral lesions due to the same cause, or to an extension to the neighboring organs, as in the case of left-sided pleurisy with pericarditis. It takes into consideration the hereditary tendencies of the patient, his age, surroundings, occupation, mode of life and habits. Diagnosis is clearly the only basis for rational therapeutics and reasonable prognosis. The medical sciences deal with diseases, the art of diagnosis with individuals. Disease is not an entity, but the sum of the phenomena of the reaction of the organism to pathogenic influences.

There are various methods of diagnosis, all of which may be included under the two general groups of DIRECT and INDIRECT DIAGNOSIS.

Direct Diagnosis.

A direct diagnosis is made when the history of the case and the clinical phenomena are sufficient to warrant a positive conclusion. The history of a violent prolonged chill, followed by high fever and pain in the chest, with cough, rusty sputum containing pneumococci, dulness upon percussion in the affected area, crepitant râles, and bronchial breathing, justify a direct diagnosis of croupous pneumonia. The previous history of the attack is not always necessary, the foregoing associated symptoms and signs being sufficient for the diagnosis of pneumonia even when the patient is delirious or too ill to give an account of himself.

The direct method is sometimes described as the semeiological method. The diagnosis is based upon the clinical phenomena of the disease and is reached by analysis and induction. When the data are adequate it is altogether the most scientific and satisfactory method.

Indirect Diagnosis.

The indirect method must be employed when the clinical phenomena are obscure or insufficient for a direct diagnosis. The results are not always conclusive and the diagnosis may remain for a time one of probability. This method includes differential diagnosis and diagnosis by exclusion.

The differential method is based upon the recognition of the essential phenomena by which one disease may be discriminated from others of a group presenting similar manifestations. A young person may present himself complaining of the following symptoms: Loss of flesh and strength, occasional irregular chills, followed by fever and sweating, shortness of breath upon exertion, cough and pain in the chest with scanty expectoration. Upon inspection the respiratory movement of the right side is diminished. The right thorax is found to be enlarged and altered in contour. There is faint cyanotic discoloration with œdema in the infra-axillary region. The heart is displaced to the left and the lower border of the liver downward. Vocal fremitus is enfeebled. There is marked dulness upon percussion over the lower part of the chest, continuous with the liver dulness, while the percussion note over the upper portion has a slightly tympanitic quality. Upon auscultation the vesicular murmur is faint and distant. Neither râles nor friction sounds are heard. The greater number of these symptoms and physical signs may be encountered in (a) abscess of the right lobe of the liver, (b) malignant disease of the pleura, (c) serofibrinous pleurisy, (d) empyema.

(a) Abscess of the right lobe of the liver is comparatively rare. There is frequently a history of dysentery or other disease of the abdominal viscera. The pus collection is rarely sufficiently large to displace the heart.

(b) Malignant disease of the pleura is likewise a rare affection. It usually develops insidiously without pain. It is not attended by chills or fever and does not displace the heart or liver until the growth has attained unusual proportions. It produces a profound cachexia and usually involves rather than compresses the lung, so that tympany in the upper part of the lung is absent and irregular patchy dulness is elicited over the seat of the growth.

(c) Serofibrinous pleurisy does not usually give rise to fever or, even when massive, to disturbance of the circulation of the wall of the chest or œdema.

(d) The essential phenomena by which, when present, empyema may be discriminated from the foregoing affections, in addition to the signs of compression of the lung and displacement of adjacent organs, are chills, fever, sweating, and cyanosis and œdema of the chest wall.

Diagnosis by exclusion differs from differential diagnosis only in its scope. It seeks to establish the nature of the disease by the negative process of showing what it is not. The various diseases presenting similar clinical phenomena are compared in turn with the case under consideration, and one after another excluded, the diagnosis of that disease being finally made to which the malady most closely conforms. In the above example we should first set aside abscess of the liver, then malignant disease of the pleura, then serofibrinous pleurisy, and by exclusion arrive at the diagnosis

of empyema. Diagnosis by exclusion is a tedious and inconvenient method, not, however, without value in difficult and obscure cases. It may be employed with advantage in clinical teaching. Other methods are:

Causal or Etiological Diagnosis. — The nature of an obscure malarial disease with or without fever may be determined by the discovery of the æstivo-autumnal parasite in the blood, or the tuberculous basis of impaired health with cough and obscure physical signs may be revealed by an examination of the sputum. When such a diagnosis concerns germ diseases it is spoken of as Bacteriological Diagnosis.

Hæmatological Diagnosis.—This may depend upon (a) the specific agglutinating properties of the serum, as in enteric fever or dysentery; (b) the morphology, as in pernicious anæmia or leukæmia; (c) the presence of parasites, as in malaria or trypnosomiasis; (d) the result of cultures; and (e) serodiagnostic reactions.

A provisional diagnosis is that which best accords with the sum of the probabilities when the data are insufficient, or pending a further investigation of the facts. Such a diagnosis may serve as a working hypothesis for therapeutic purposes and the general management of the patient. It can be revised or confirmed.

A surgical diagnosis is made from the stand-point of the surgeon, and may in proper cases be confirmed or set aside during the life of the patient by an exploratory operation.

Functional diagnosis is the determination of the degree of the impairment of the functions of organs caused by local affections or the extent of the interference with physiological processes resulting from general disease, and the bearing of such impairment or interference upon the future of the individual as regards health and prolongation of life. Functional diagnosis is closely allied to prognosis.

A therapeutic diagnosis is that procedure by which in obscure cases the nature of the disease is determined by the results of treatment. This method is of very limited application. A provisional diagnosis of malaria having been reached by the process of exclusion, the patient may be put at rest and quinine administered in proper doses. Should the symptoms promptly disappear, the diagnosis of malaria becomes probable. A similar diagnosis of syphilis having been reached by analogous methods, the subsidence of symptoms upon the administration of mercurials or the iodides may in some cases confirm the diagnosis. In almost all such cases there are other and better methods of diagnosis which may be employed concurrently with the treatment. In grave or urgent cases it is, however, better to give the patient at once the benefit of the doubt.

Clinical diagnosis is the diagnosis made at the bedside.

Anatomical diagnosis is the diagnosis made by the pathologist in the post-mortem room.

It is not in all cases possible to make a positive diagnosis at once. Time may be required for a more thorough investigation of the history of the case, a closer study of the patient's surroundings, repeated observation, or for the report of examinations conducted in the clinical laboratory. Information bearing upon the previous history of the patient or the beginning of his illness cannot always be obtained. He may be delirious,

unconscious, or may have lost the power of speech. The history communicated by his friends is often uncertain and misleading. Persons of the lower classes are very commonly indifferent to symptoms which are not painful or disabling and lack the ability to describe their sensations. Many persons, on the other hand, often intentionally, sometimes unconsciously, make false statements in regard to their past life and present symptoms. Some parts of the narrative are exaggerated, others suppressed. Symptoms may be imitated and superficial lesions artificially produced. Hence a group of feigned diseases, against which the physician must be upon his guard.

Malingering.—The term malingerer is used to describe one who intentionally simulates a disease. Malingering occurs in every grade of life and under various circumstances. It is to be suspected when a simulated disease lacks essential symptoms or its picture is overdrawn, and when there is lack of correspondence between the alleged symptoms and the actual signs or the obvious general health; it is to be detected by close study of the case under various conditions, by the use of instruments of precision, and in some cases by the application of powerful faradic currents or an examination under anæsthesia. The over-indulged child, to avoid his lessons or escape punishment, may feign an illness; an older person, to excite compassion or from mere love of deception. It is common among beggars, sailors and soldiers, those improperly seeking pensions, and claimants against corporations for accidental damages. The simulation of disease is, however, not always intentional. Hysterical and neurasthenic individuals sometimes exaggerate symptoms or imitate the manifestations of disease without purpose or intention—the unconscious mimicry of disease. There are those, on the other hand, who from motives of delicacy or shame, or in consequence of natural reserve, or from fear of having their apprehensions confirmed, refuse to consult the physician, or when forced to do so give a garbled and incomplete history of their sickness. This may occur among those suffering from venereal diseases or chronic diseases popularly regarded as incurable, as tuberculosis and cancer.

The diagnosis of an obscure case occasionally demands an investigation of the surroundings of the patient at the time of the development of the illness. Time may be required to ascertain etiological conditions relating to his food, drink, occupation, endemic influences, or exposure to transmissible diseases locally epidemic. Questions of this kind frequently arise at a period like the present, when facilities for commercial intercourse are increasing and when military operations and the exigencies of trade have greatly extended travel to all parts of the world.

Repeated examinations may be necessary in order to obtain accurate impressions when the physical signs are obscure or ill defined. Excessive subcutaneous fat, local œdema, or general anasarca may interfere with the physical exploration; or local tenderness, intense pain, great restlessness, or an unwillingness on the part of the patient to submit to an examination may give rise to delay. In other cases the unusual character of the symptoms or an association of clinical phenomena not previously encountered may render repeated examinations necessary. During the stage of invasion in the acute febrile infections a positive diagnosis is often impossible.

The advances of modern medicine have enormously increased our knowledge of diseases and the precision of diagnosis. In all departments of clinical medicine, scientific accuracy has taken the place of probability. The every-day routine examinations of the clinical laboratory cannot be made off-hand. The more elaborate investigations involved in obscure cases demand technical skill and a reasonable time. The reports are necessary to a final diagnosis. An immediate diagnosis is not only not necessary, it is very often not possible. Haste involves the risk of error. Conclusions cannot be reached until the premises are established. A provisional diagnosis may serve to meet the immediate requirements of the situation. Treatment may be instituted in response to urgent indications. When in the period of invasion of an acute illness there is reason to suspect a transmissible disease, such as scarlatina or variola, the same measures of prophylaxis should be instituted pending the evolution of the process that would be employed if the suspected disease were actually present.

There are cases in which diagnosis in a broad sense is impossible. A name may be given to some prominent symptom or group of symptoms, but the essential pathological process may remain obscure until its nature is revealed upon the post-mortem table.

When possible a positive diagnosis should be made at once; in all cases as soon as practicable. The student is, however, warned against making any but a provisional diagnosis upon insufficient data. To ask for delay is by no means a confession of ignorance; on the contrary, it is the course dictated by knowledge and experience. Intelligent people, who seek the best professional advice, fully understand this. It is only the ignorant who are satisfied with a phrase for diagnosis, a prescription dashed off at sight and no directions whatever, who insist upon being told what is the matter at once.

II.

MEDICAL TOPOGRAPHY.

Medical topography is that branch of diagnosis which has for its object the consideration of the boundaries and relations of the external parts and internal organs of the body. Various points, lines, and regions or areas, some artificial, others natural, serve the purposes of this method of clinical investigation.

THE HEAD.

The head is divided by anatomists into two parts, the cranium and the face.

The Cranium.

The skull encloses and protects the brain. It is divided into regions corresponding with the superficial bones which enter into the formation of the skull,—namely, occipital, parietal, frontal, and temporal. These regions are separated by the cranial sutures. Opposite the angles of the parietal bones are spaces called fontanelles,—*fons*, a fountain,—which remain unossified after the bony growth of the skull is elsewhere completed. Of these, two in the median line, the anterior and posterior fontanelles, are important.

The regions of the skull serve for the localization of subjective sensations, as pain or headache, and superficial lesions, as craniotabes, nodes, nævi, injury, or suppuration. The mastoid process of the temporal bone is an important landmark, as indicating the extension of middle-ear disease. The greatest convexity in the frontal region on either side is known as the frontal eminence. It is separated by a slight depression below from the superciliary ridge, at the level of which in the median line is the nasal eminence or glabella. About the inner third of the orbital arch is the supra-orbital notch or foramen, a point of tenderness in supra-orbital neuralgia.

Sutures.—Failure on the part of the cranial bones to unite, with persistent wide sutures, may be due to hydrocephalus, cretinism, or in very rare instances to antenatal rickets.

Fontanelles.—Variations in Prominence.—Bulging of the fontanelles is a common symptom in infants and young children. It is much more marked in the anterior fontanelle. When persistent it indicates organic diseases of the brain, as hydrocephalus, meningitis, or intracranial hemorrhage, which is in infants far more commonly meningeal than cerebral. When transient it is usually pulsating and associated with high temperature and other symptoms of an acute febrile infection.

Retraction of the fontanelles occurs in chronic wasting diseases, as tuberculosis, infantile atrophy or marasmus, and colitis, and in acute diarrhœal affections, as enterocolitis and cholera infantum.

Variations in Size.—The posterior fontanelle is normally obliterated about the sixth week. The anterior remains patulous as at birth or even slightly increases in size up to about the ninth month, and closes before the end of the second year. Delay in closing beyond this period is commonly associated with wide and ununited sutures and occurs in rickets and hydrocephalus. The diameter of the anterior fontanelle at the end of the first year is normally about 2.5 centimetres. A greater width occurs in rickets and some cases of congenital syphilis. A very wide fontanelle is characteristic of hydrocephalus.

The Face.

The regions of the face are the orbital, nasal, buccal, and oral. They contain the muscles of expression and are of great importance in the diagnosis of local and constitutional disease, as well as in the recognition of mental and emotional conditions. The facies in various conditions will be described in a later chapter. Changes caused by nervous and ocular disorders will be considered under their appropriate headings.

DEFORMITIES OF THE HEAD IN THE NEWBORN.

Caput Succedaneum.—A swelling of the scalp caused by pressure during parturition. The lesion consists of passive congestion with extravasation of blood and œdema of the tissues of the scalp at the area of absence of pressure, namely, the part of presentation. The tumor is irregularly circumscribed and does not fluctuate. It disappears without treatment in the course of a few days. This condition is to be distinguished from—

Fig. 1.—Caput succedaneum. Male, 2 hours old.—Rotch.

Cephalhæmatoma.—A tumor formed during labor by hemorrhage into the space between (a) the occipitofrontalis aponeurosis and the periosteum, or between the periosteum and the skull—external cephalhæmatoma—or (b), between the skull and the dura mater—internal cephalhæmatoma.

(a) External Cephalhæmatoma.—The most common variety is subperiosteal. It occurs in the form of an irregular, circular, flat tumor over one or, in rare instances, both parietal bones. There is distinct fluctuation, but the overlying skin is not discolored. Slight elevation of the bone at the border of the swelling may be felt in a few days, with obscure crepitus. The condition is to be distinguished from caput succedaneum by its location, fluctuation upon palpation, and the examination of fluid withdrawn by aspiration. The bony rim is diagnostic at a later period. It is not to be confounded with a depressed fracture, which is irregular in outline and lacks the distinct tumor formation with fluctuation and the rim-like bony circumference characteristic of hæmatoma.

(b) Internal Cephalhæmatoma.—A very rare condition which ends in the death of the child. There are pressure symptoms. It is sometimes associated with the external form. It has occurred in breech presentations.

Fluctuating tumors arising from the course of the cranial sutures are usually situated in the occipital region or at the glabella. Three varieties are described.

Meningocele.—This term is used to designate a hernial protrusion of the meninges through an opening in the bony cranium resulting from defective ossification or failure in suture formation. It may result from intra-uterine hydrocephalus. The tumors usually contain cerebrospinal fluid, and are translucent, with large veins upon the surface. In some instances an impulse may be felt upon crying and the tumor can be reduced by gentle pressure.

Fig. 2.—Hydro-encephalocele.—Rotch.

Encephalocele.—This form of cerebral hernia is more common. The tumor contains brain substance in addition to the membranes.

Hydro-encephalocele. — The hernial contents consist of the membranes, brain tissue surrounding one of the ventricles, and a portion of the ventricle itself distended with cerebrospinal fluid. These tumors vary in size from a walnut to a large orange and tend to increase in size. They are usually pedunculated. The prognosis is unfavorable, though remarkable recoveries have occurred after operation.

Anencephalia.—This developmental defect is rarely complete. Partial anencephalia is the usual form. In accordance with a recognized pathological law, the deficiency of contents causes microcephalic deformity of the skull.

Hydrocephalus.—Congenital internal hydrocephalus is a common cause of deformity of the skull in the newborn. The head is markedly enlarged; the cranial bones are thinned and displaced outwards; the

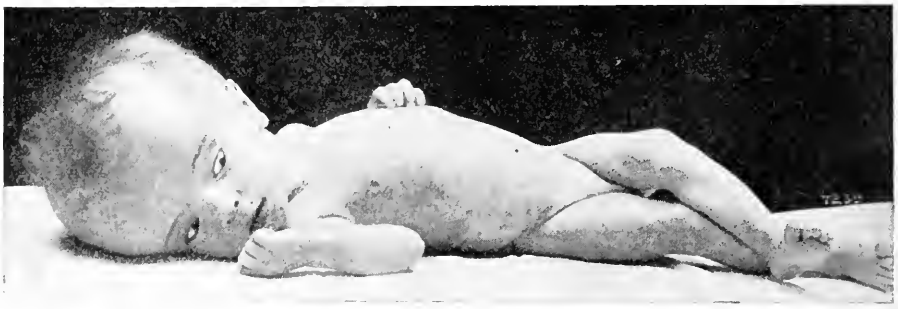


Fig. 3.—Congenital internal hydrocephalus. Male, 7 months old.—Rotch.

sutures widely separated and the fontanelles prominent and fluctuating. In marked cases the temporal and parietal bones flare outward so that the cranium is more or less pear-shaped, the greatest diameter being in the upper part. The face is usually normal in size, but it looks abnormally small, being dwarfed by the great size of the head.

THE NECK.

Length and Thickness.

In early infancy the neck appears short on account of the large size of the head and its tendency to fall forward, and the relatively high position of the sternum and clavicles. The neck appears to be broad in comparison with its length also by reason of the large amount of subcutaneous fat. In fact at all periods of life the thick neck of obese persons appears short, an appearance heightened by the accumulation of fat known as the double chin.

A short thick neck and stout plethoric body constitute the chief structural factors in the so-called habitus apoplecticus. On the other hand, a long slender neck with a prominent larynx, and narrow flat chest with projecting scapulæ, are characteristic of the habitus phthisicus. But both these designations are misleading, since apoplexy is dependent upon a condition of the arteries and frequently occurs in spare persons with long thin necks, and pulmonary tuberculosis is the result of infection and not rarely selects its victims among those who have well-formed chests and necks, and occasionally among those who are stout, with short thick necks.

Contour.

Larynx.—In lean persons the larynx is prominent and forms the projection anteriorly in the median line known as the Adam's apple. In fat persons this organ is much less noticeable. Descent of the larynx upon inspiration occurs in all forms of severe dyspnœa, and especially in the spasmodic respiration which often precedes death in respiratory diseases attended with stenosis of the larynx or œdema, collapse or extensive consolidation of the lungs. Pressure displacements of the larynx and trachea sometimes result from the presence of aneurismal or other tumors of the neck. They are usually lateral. Some degree of lateral displacement may also result from pleural adhesions and the traction of a contracting lung in neglected pleurisy or fibroid phthisis. Moderate bilateral prominence and enlargement of the neck without spastic contraction occur in the habitual dyspnœa of severe chronic bronchitis, emphysema, bronchial asthma, cardiac disease and certain cases of chronic uræmia—so-called renal asthma. Rigidity of the neck is sometimes due to myalgia of the cervical muscles, spondylitis deformans involving the cervical vertebræ, or caries. It may be caused by painful inflammatory processes, as acute adenitis, parotid bubo, mumps, boils, or carbuncles.

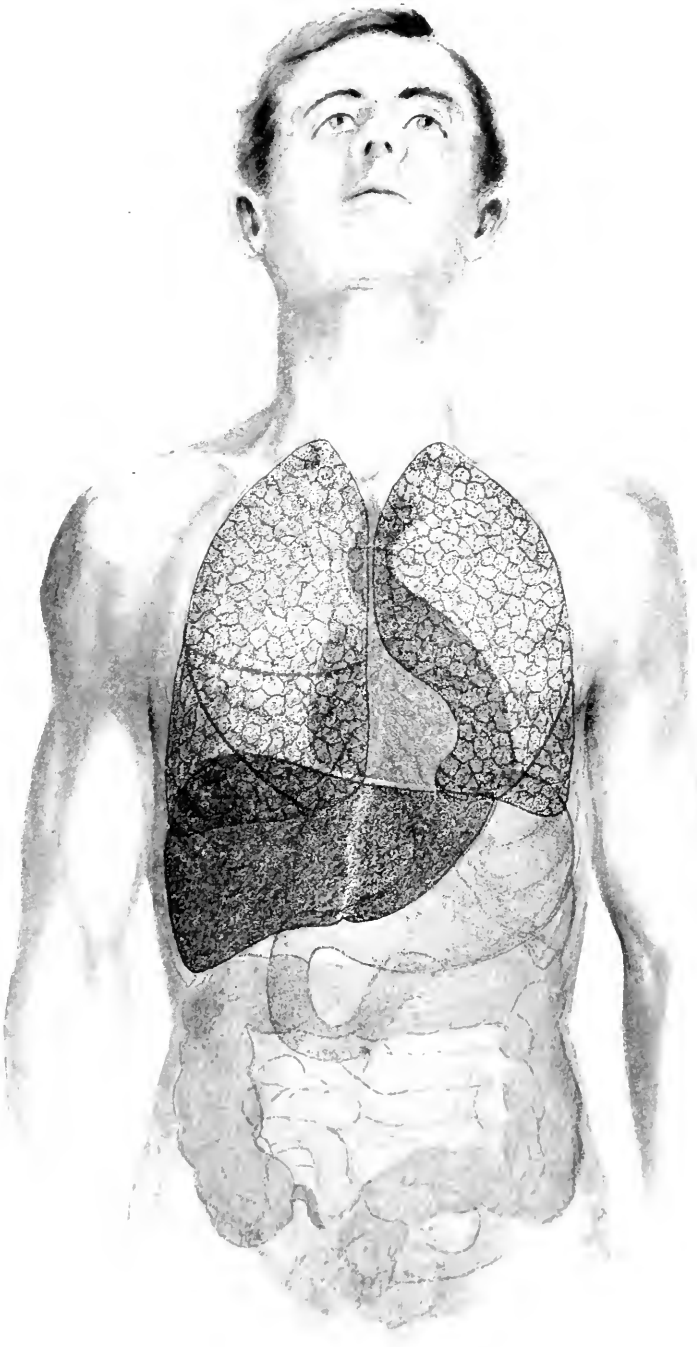
Thyroid Body.—This gland is situated in the lower part of the neck and embraces the trachea in its upper part, reaching up to the larynx on each side. It consists of two lateral lobes united by an isthmus. The right lobe is usually slightly longer and wider than the left. Both the larynx and the thyroid body which is in relation with it rise with the act of swallowing. Enlargement of the thyroid body usually affects the isthmus and both lobes, but one—very often the right—to a greater extent than the other. The enlargement may be vascular, parenchymatous, fibroid,

cystic, or due to adenoma, carcinoma, tuberculosis, or gumma. Vascular enlargement of the thyroid body may be physiological, occurring during menstruation or pregnancy and subsiding at the termination of these events, or pathological, as in exophthalmic goitre, when it is often variable in size and attended with marked pulsation, thrill, and murmur. Venous hyperæmia may be due to the pressure of an aneurism or mediastinal tumor. Parenchymatous enlargement or simple goitre may be of moderate size, but in some instances attains enormous dimensions, protruding beyond the chin and hanging over the sternum. Cystic goitre when multiple may be recognized by the smooth, hemispherical, close set, elastic nodules upon the surface; when single and larger, by fluctuation. Thyroid abscess is rare and usually accompanied by local inflammatory œdema and grave constitutional symptoms. Cancer, tuberculosis, and gumma rarely involve the thyroid and may be recognized by their local characters and the associated constitutional phenomena. An underlying aneurism or mediastinal growth may displace the thyroid upwards and forwards or to either side. In aneurism of the innominate, the displacement is towards the left. An aneurism sometimes imparts its movements to the overlying thyroid. Atrophy of the thyroid may give rise to flattening of the surface. More commonly it can be recognized only upon palpation. It is usually accompanied by the symptoms of cretinism or myxœdema.

Fig. 4.—Cystic goitre.

Muscles.—One or both sternomastoid muscles may be hypertrophied and prominent. In torticollis or wry-neck the contraction is usually unilateral, and the neck is rotated so that the mastoid is drawn towards the inner end of the clavicle, the chin raised and the face turned towards the unaffected side. In rare cases wry-neck is bilateral.—retrocollic spasm, —the head retracted and the face turned upward. The spasm in both forms of torticollis may be tonic or clonic. The disease is sometimes congenital.

The Clavicles.—The position of these bones has much to do with the appearance of the neck as regards length. They are high in deep-chested persons with large lungs and in emphysema; low in flat-chested individuals with small lungs and in phthisis and pulmonary fibrosis from any cause. These bones are deformed after fracture and sometimes present nodes and irregularities of the surface caused by syphilitic periostitis. Prominence in the retroclavicular space sometimes occurs in emphysema



General anatomical outlines and relations of the thoracic and abdominal organs.

of high grade, but as a rule the clavicles in this condition are prominent and both the retro- and infraclavicular spaces are retracted. The subcutaneous fat pads of myxœdema are frequently seen above the clavicles and sometimes at the root of the neck posteriorly. The neck is occasionally the seat of extensive inflammatory œdema with violaceous discoloration—collar of brawn—especially in scarlet fever, erysipelas, and infected wounds, and sometimes much distorted by subcutaneous emphysema, such as follows rupture of the pleura or wounds or operations involving the upper air-passages.

THE THORAX.

The thorax is of conical shape with convex walls. Its truncated upper end is narrow and bounded by the first dorsal vertebra, the first pair of ribs, and the manubrium of the sternum. Its expanded base is filled in by the vault of the diaphragm. The anterior border of the base curves downwards and backwards on each side from the xyphoid cartilage to the twelfth rib. Its transverse diameter greatly exceeds its anteroposterior diameter, which is further shortened in the middle line by the projection of the spinal vertebræ forwards into the cavity of the thorax. This space contains the heart and great vessels together with the pericardium, the lungs and pleuræ, the trachea, the greater part of the œsophagus, and the thymus gland or its remnant.

Anatomical Landmarks of the Thorax.

ANTERIOR SURFACE.

The Chest.—The clavicles, sternum, ribs, and interspaces constitute natural surface conformations to which, for purposes of study and description, clinical phenomena may be referred.

The Clavicles.—The part immediately above these bones on either side is known as the supra- or retroclavicular space; that immediately below them as the infraclavicular space. Into the supraclavicular spaces the apex of the lung enters to a slight extent, usually a little further on the right than on the left side. In well-developed lungs these spaces are not retracted, but in ill-developed lungs and in pathological states characterized by contraction of lung tissue they are more or less strongly depressed.

The Sternum.—The upper border of this bone is marked by a large incurvation known as the episternal notch, which limits the root of the neck anteriorly and in which can be felt at times the pulsating aorta—dilatation, dynamic pulsation, aneurism of the transverse arch. At the line of juncture of the manubrium and gladiolus or body is a more or less prominent transverse line or prominence, better developed in the male—the angle of Ludovicus. At the lower end is the xyphoid or ensiform appendix, variable in size and shape and sometimes having its tip everted in such a manner as to form an infrasternal depression or fossa.

The Ribs and Intercostal Spaces.—The Ribs.—In spare persons the ribs may be counted with ease. When, however, there is much subcutaneous fat, the recognition of any particular rib is sometimes difficult. The first rib may be known by the articulation of its cartilage with the

sternum at a point immediately below the articulation of the clavicle. The articulation of the second costal cartilage is directly opposite the junction of the first and second pieces of the sternum—angulus Ludovici. The ribs slope downwards from their spinal articulations in such a manner that their chondrosternal articulations lie at a much lower level, the articulation of the first rib anteriorly being in quiet breathing on the horizontal plane of the fourth rib at the back and so on to the seventh rib. In the expiratory type of chest this oblique position of the ribs is somewhat increased; in the inspiratory type it is much diminished.

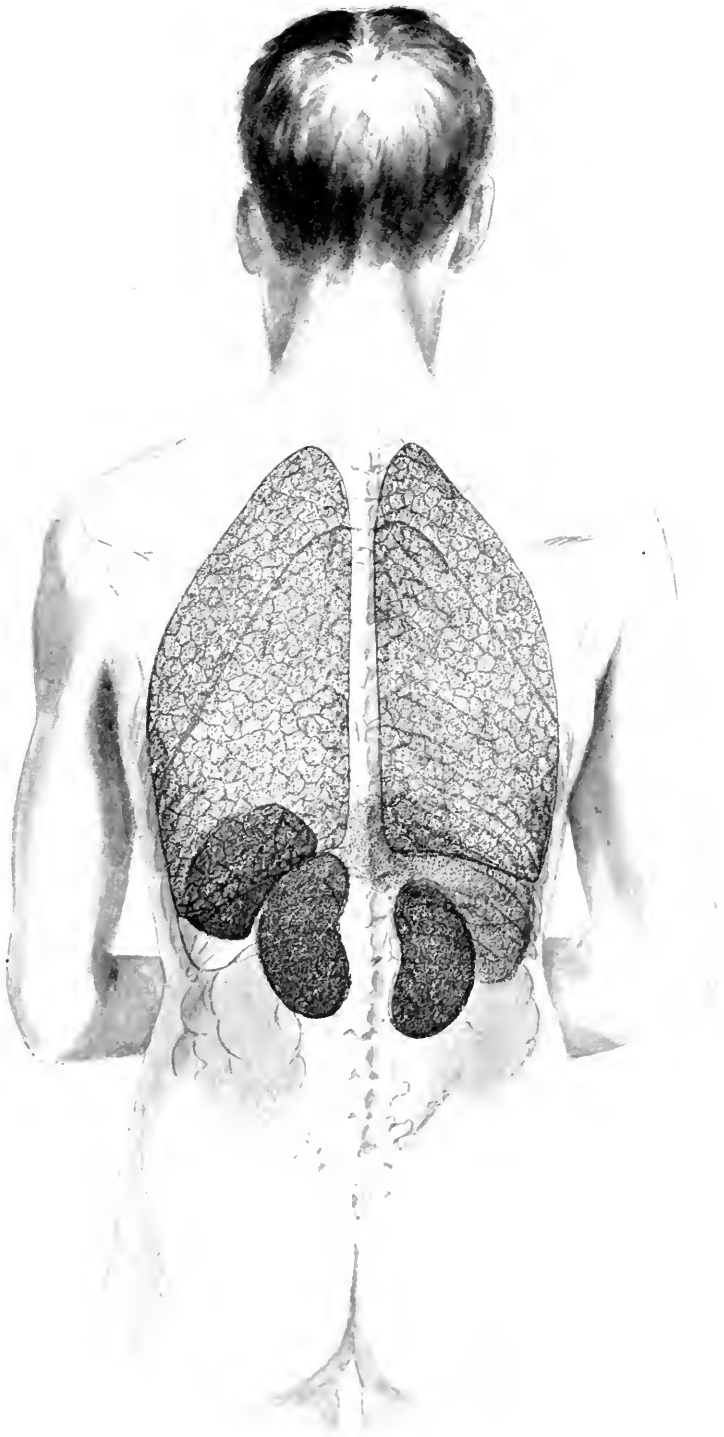
The Intercostal Spaces.—These spaces correspond to the ribs and cartilages immediately above them—that is, the first space lies immediately below the first rib. They are wider in front than behind. In expiration the upper spaces are increased in width and the lower narrowed, while in inspiration these conditions are relatively reversed. It is in accordance with this fact that the upper spaces are wider and the lower narrower in the expiratory, while the upper are narrower and the lower wider in the inspiratory form of chest. In fat persons the intercostal spaces cannot be made out, but in those who are lean they appear as shallow, parallel, oblique furrows symmetrically arranged upon each side of the chest. They are deeper upon inspiration than on expiration or quiet breathing, and conspicuously so in obstructive dyspnœa. These furrows are obliterated in massive pneumonia and in pleural effusions and the spaces may actually bulge in old cases of large empyema. Local protrusion of the chest wall such as occurs in large hypertrophy of the heart in early life causes widening of the overlying intercostal spaces. The unilateral flattening of the chest which accompanies fibroid phthisis or follows a neglected pleural effusion, crowds the ribs together, even in some instances to overlapping, and in this manner obliterates the spaces in whole or in part.

Normal Cardiac Pulsation.—The apex beat is seen in the fifth intercostal space to the left of the parasternal line, while undulatory pulsation in several spaces occurs in dilatation of the heart, and heaving pulsation over a large area in marked hypertrophy of that organ. In rare instances intercostal pulsation is due to a neglected empyema. The pulsation is almost always in the anterolateral aspect of the chest upon the left side. In mitral and aortic stenosis, aortic insufficiency, cases of congenital malformation of the heart, and aneurism of the aorta, thrills may be felt upon palpation.

The Nipple.—This organ is not without value as a topographical landmark in children and spare males, but in women and obese persons of both sexes its position is extremely variable. When there is little fat and the mammæ are undeveloped the nipple is situated about the fourth intercostal space, sometimes over the fourth, sometimes over the fifth rib, and in a vertical line intersecting the middle of the clavicle—the mammillary line. It is obvious that the nipple—mammilla—is not a satisfactory anatomical landmark. The midclavicular line is much more useful.

POSTERIOR SURFACE.—THE BACK.

The Spine.—In children and lean persons the spinous processes are prominent. In muscular adults and fat people they are situated in the middle of a shallow longitudinal groove formed by the prominence of the



General anatomical outlines and relations of the thoracic and abdominal organs.

erector spinæ muscles on either side. They become more prominent when the patient bends strongly forward. Owing to the denseness of the overlying musculotendinous tissues, the spines of the upper five cervical vertebræ cannot as a rule be recognized upon palpation. The sixth may be felt and seen in many persons, and the seventh—vertebra prominens—is usually conspicuous and forms a point of departure from which the thoracic and lumbar spines may be counted. The eighth and ninth thoracic spines are normally somewhat more prominent than the others. Marked prominence of one or more vertebral spines, with tenderness upon pressure and pain upon rotary movements of the spine or jarring, is significant of spinal caries, usually tuberculous, rarely syphilitic. There is frequently angular curvature.

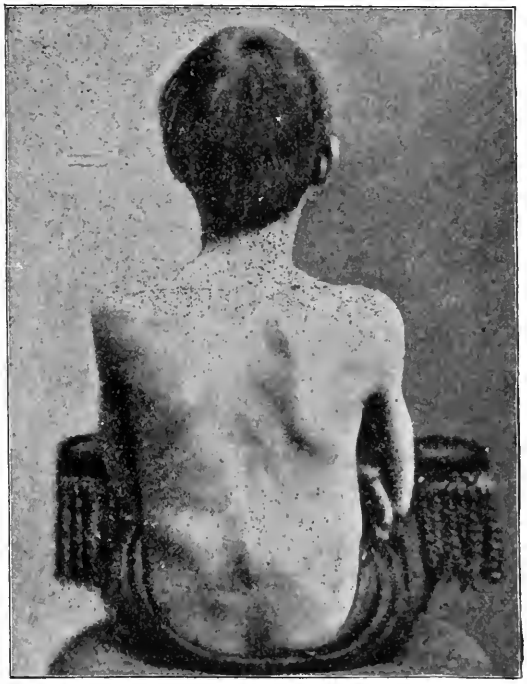


Fig. 5.—Spinal caries. Lumbar region.—Young.

Kyphosis.—The curvature is in the sagittal plane with the concavity anterior. It is chiefly thoracic, sometimes cervicothoracic, and may constitute nothing more than one of the skeletal changes due to old age. It occurs also in those who habitually carry heavy burdens on the head and shoulders, in emphysema, rickets, osteitis deformans, and acromegaly. This rounded curvature is to be distinguished from the sharper, often angular curvature of vertebral caries or mollities ossium.

Scoliosis. — A rotary-lateral curvature usually involving the upper thoracic spine with compensating curvature in the lower thoracic and lumbar regions. Less commonly it affects the cervical or lumbar regions. Scoliosis is very common in school-girls in consequence of poor muscular development and faulty desk attitudes. It may result from the habitual carrying of heavy weights on the same arm, inequality in the length of the legs, deformity of a foot, tilting of the pelvis, old sciatica, the arrested growth of a limb following infantile palsy, hemiplegia, and mollities ossium. The deformity of the chest following long-neglected pleural effusion, serofibrinous or purulent, includes dorsal scoliosis, the concavity looking towards the affected side.

Lordosis.—An exaggeration of the normal lumbar curve occurs in advanced pregnancy, large abdominal tumors and ascites, progressive muscular atrophy, and pseudohypertrophic muscular paralysis. The attitude

in the last condition is characteristic. The legs are separated, the head is thrown back, the spine strongly curved, and the abdomen thrust forward.

Spina Bifida.—This is a developmental fault consisting of failure on the part of the laminæ of the vertebræ to unite. The usual site is in the lumbar or lumbosacral region. The protruding tumor is in the middle line, sometimes covered with normal skin, sometimes with a thin, translucent membrane. There are two varieties:

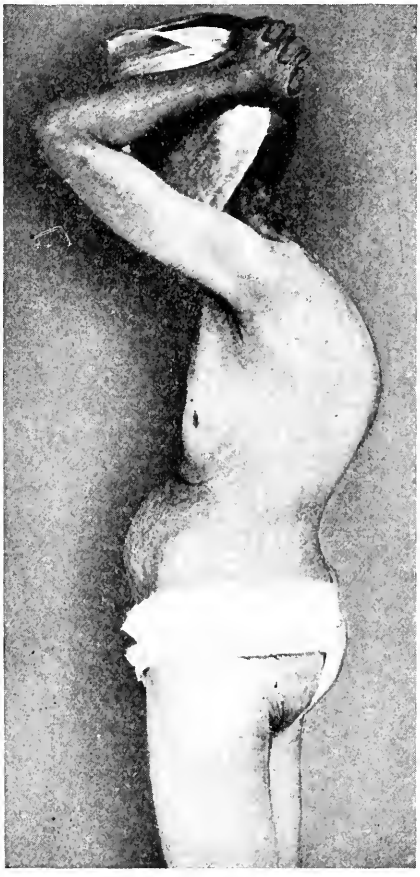


FIG. 6.—Kyphosis.

FIG. 7.—Senile kyphosis.

SPINA BIFIDA OCCULTA, in which the sac is walled off from all connection with the spinal canal, and—

SPINA BIFIDA VERA, in which the cyst is filled with cerebrospinal fluid and increases in size during violent crying, and can be diminished by gentle pressure.

Three subvarieties are recognized:

Spinal Meningocele.—The protruding membranes contain only cerebrospinal fluid.

Meningomyelocele.—The sac contains not only fluid but also substance of the cord. This is the most common form.

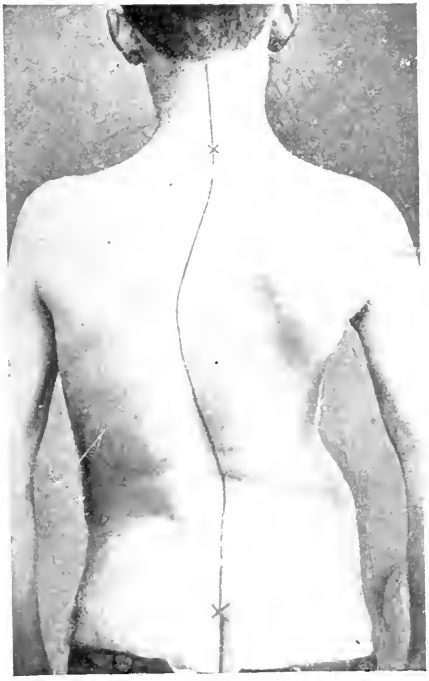


Fig. 8.—Primary left dorsal scoliosis.—Young.

Fig. 10.—Spina bifida of lumbar region. Male, 5 years old.—Rotch.

Fig. 9.—Extreme lordosis in progressive muscular atrophy.—Young.

Syringomyelocele.—The sac is formed of the membranes and a protruding portion of the cord, the central canal being dilated to form the cavity of the tumor.

This group of deformities is commonly associated with other developmental defects. Exceptionally spina bifida occurs in children otherwise healthy and well developed.

The Scapulæ.—These flat, triangular, trowel-like bones are placed symmetrically upon the upper and back part of the thorax and extend, when the arms hang by the sides in the erect posture, from the second to the seventh ribs. They are attached to the skeleton by the clavicle and the humerus and are therefore freely movable. When the arms are folded and the body is bowed forward, the interscapular space is much increased, an important fact in physical diagnosis. The inner borders of the scapulæ project in consequence of muscular weakness, palsy, and changes in the contour of the chest. Combinations of these causative conditions may occur in the same case. Both inner borders project in the alar or pterygoid chest and in the progressive muscular dystrophies affecting the shoulder girdle. The abnormal mobility of the shoulder-blades arising from loss of muscular tone permits the inner borders to project like budding wings. The inner border stands out upon the affected side in contraction of the chest from pulmonary fibrosis; in associated serratus and trapezius paralysis, especially when the arms are held out in front in the horizontal plane; in scoliosis due to various causes, and sometimes upon the left side in large aneurism of the descending portion of the arch of the aorta.

Immobility of the Spine.—Flexion, extension, and lateral and rotary movements may be restricted or wholly prevented by various pathological conditions, as (1) those giving rise to pain in movement, among which the more common are traumatism, myalgia—lumbago—abscess, carbuncle, meningeal hemorrhage; (2) those involving spasm, as cerebrospinal fever and the spastic form of myalgia; (3) those affecting the joints and bones, most of which terminate in ankylosis, as traumatic, gonorrhœal, or tuberculous disease and spondylitis deformans; and (4) certain neuroses, as many of the cases of so-called typhoid spine, railway spine, hysterical spine, irritable spine, and so on.

LATERAL SURFACES.

The landmarks are the axilla—armpit—above, the anterior and posterior axillary folds, the ribs and interspaces and the upper border of hepatic, on the right, and of splenic dulness on the left side, below. Enlarged lymph-nodes, which frequently undergo suppuration or may be tuberculous, carcinomatous, leukæmic or pseudoleukæmic, are common in the axillary space.

Artificial Lines and Spaces of the Thorax.

The following conventional imaginary lines and spaces serve a useful purpose in the examination and description of thoracic lesions. For convenience of demonstration the lines may be marked upon the surface with a dermatographic pencil. The subject is in the erect posture with his arms symmetrically disposed.

A. Vertical Parallel Lines. — With the exception of the first and last they are double—bilateral.

(a) The mesial or midsternal line.
(b) The line of the sternal border.
(c) The parasternal line, midway between the line of the sternal border, and—
(d) The midclavicular line, sometimes spoken of as the mammillary line because in individuals with undeveloped mammæ it passes through or near the nipple.

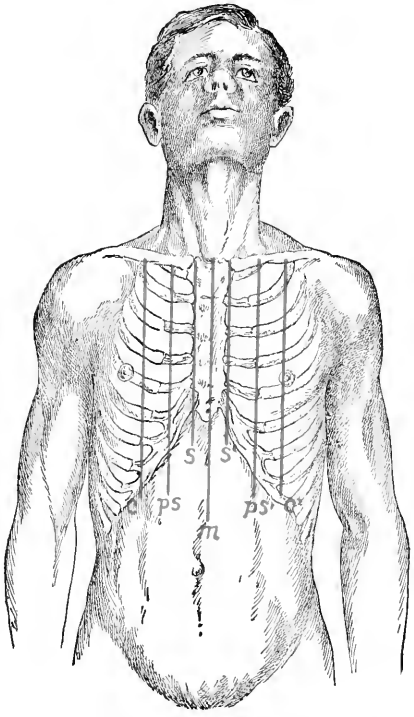


Fig. 11.—Lines of reference: Anterior.—*m*, middle line; *s*, *s'*, right and left lines of the sternal border; *ps*, *ps'*, parasternal lines; *c*, *c'*, midclavicular or mammillary lines.

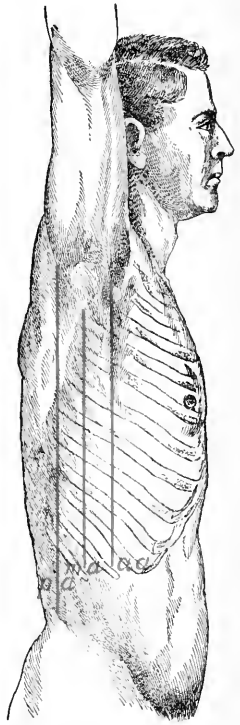


Fig. 12.—Lines of reference: Lateral.—*aa*, anterior axillary line; *ma*, midaxillary line; *pa*, posterior axillary line.

(e) The line of the anterior axillary fold.
(f) The midaxillary line.
(g) The line of the posterior axillary fold.
(h) The scapular line, passing vertically through the inferior angle of the scapula—a very movable and uncertain landmark.
(i) The posterior mesial line, corresponding to the line of the spinous processes.

B. Horizontal Parallel Lines.—These are anteriorly:

(a) A line touching the lower border of the cricoid cartilage.
(b) A line passing through the clavicles.
(c) A line passing through the third chondrosternal articulation.
(d) A line passing through the sixth chondrosternal articulation.

And posteriorly:

(a) A line touching the upper border of the scapulæ.
(b) A line passing through the spines of the scapulæ.
(c) A line passing through the inferior angles of the scapulæ.
(d) A line touching the upper border of the spine of the twelfth dorsal vertebra.

Regional Divisions of the Thorax.

By the intersection of certain of the above-described lines the following arbitrary regions are formed:

(a) The Suprasternal Region. — This region overlies the thyroid body, the trachea, and more deeply the œsophagus. The transverse aorta, when dilated, extends into it and may be felt pulsating above the level of the sternal incisura.

(b) The Upper Sternal Region. — Beneath the breastbone lie the remnants of the thymus, the mesial borders of the upper lobes of the lungs, and more deeply the transverse arch of the aorta.

(c) The Lower Sternal Region.—Within the limits of this space lie the mesial border of the right lung, the termination of the fissure forming the upper boundary of the middle lobe, and that part of the right heart which constitutes the area of superficial dulness.

On each side:

(d) The Supraclavicular Region.—This space lies above the upper edge of the collar-bone and contains the apex of the corresponding lung.

(e) The Clavicular Region.—A space of no great moment in diagnosis. It corresponds to the boundaries of the inner half of the bone. The clavicle may be used as a plexímeter in direct percussion.

(f) The Infraclavicular Region.—A most important area of the chest. It is bordered above by the line of the clavicles, internally by the line of the sternal border, externally by the line of the anterior axillary fold projected upward to the acromion process, and below by the horizontal line passing through the third chondrosternal articulation. It contains on either side that part of the upper lobe of the lung in which tuberculous bronchopneumonia is as a rule first recognizable.

(g) The Mammary Region.—From the lower border of the preceding to the line passing through the sixth chondrosternal articulation. This space contains on the right side a part of the upper and middle lobes and the fissure separating them, together with the right auricle near the sternal border, and more deeply in the vault of the diaphragm the convexity of the right lobe of the liver. It overlies on the left side the extrasternal area of superficial dulness, the apex of the right and of the left ventricle, and the mesial border of the left lung with the lingula. Into the mammary region on each side extend the interlobar fissures of the lungs.

(h) The Inframammary Region.—This area, which extends from a horizontal line through the sixth chondrosternal articulation downwards, overlies the liver on the right side, and upon the left a portion of the left lobe of the liver, the fundus of the stomach, the transverse colon, and the spleen. On the left is Traube's semilunar space.

(i) The Axillary Region.—This space is bounded by the lines of the axillary folds and the armpit above. It is a diagnostic territory of some importance.

(j) The Infra-axillary Region.—The upper boundary is the line which passes through the sixth chondrosternal articulation; its lower is the base of the chest. In this region, upon the left, the upper border of splenic dulness may be demonstrated upon percussion. The interlobar fissure traverses the axillary and infra-axillary spaces.

(k) The Suprascapular Region.—An area of importance on account of the early manifestations of phthisis.

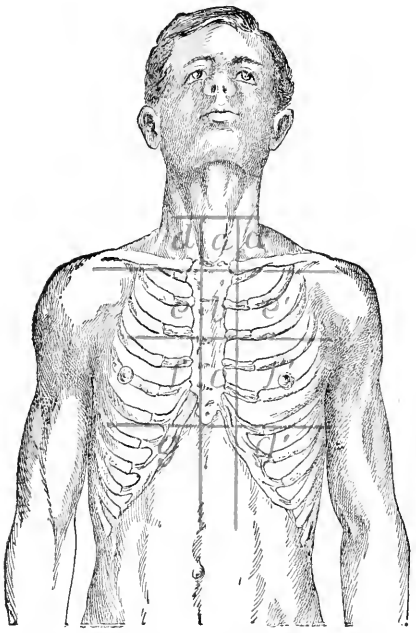


Fig. 13.—Regional divisions of the thorax: Anterior.—*a*, suprasternal region; *b*, upper sternal; *c*, lower sternal; *d*,*d'*, right and left supraclavicular; *e*,*e'*, right and left infraclavicular; *f*,*f'*, mammary; *g*,*g'*, inframammary.

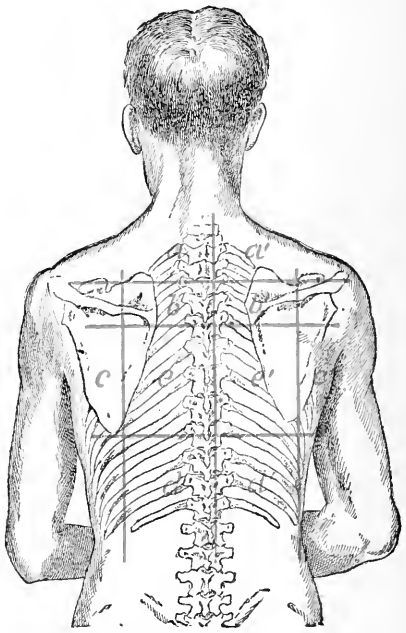


Fig. 14.—Regional divisions of the chest: Posterior.—*a*,*a'*, supraclavicular regions; *b*,*b'*, supraspinous; *c*,*c'*, infraspinous; *d*,*d'*, infrascapular; *e*,*e'*, interscapular regions.

(l) The Supraspinous Region.—That space lying between the upper border of the scapula and the spine of the scapula, and occupied by the thick supraspinous muscle.

(m) The Infraspinous Region.—From the spine of the scapula to the level of the inferior angle. The infraspinous and infrascapular regions are traversed by the interlobar fissures. This fact is of importance in the recognition of the signs of the extension of a tuberculous infiltration to the apex of the lower lobe.

(n) The Infrascapular Region.—From the angle of the scapula, namely, about the level of the seventh rib, to the base of the chest.

(o) The Interscapular Region.—The space lying between the inner borders of the two scapulæ. It extends across the spinal column and is much widened when the arms are folded and the body bent forward.

THE ABDOMEN.

The abdomen is the great cavity of the body extending from the diaphragm above to the levator muscles of the anus below. It is subdivided by an oblique plane at the brim of the pelvis into two portions, the abdomen proper and the pelvis. For the purpose of exact reference to the position and condition of the organs contained in the cavity of the abdomen in health and disease, certain lines, as in the case of the thorax, are recognized upon the surface. These dividing lines are natural, and artificial or conventional.

The Natural Lines of the Abdomen.

(a) THE LINEA ALBA in the middle line from the ensiform cartilage to the symphysis pubis.

(b) THE LINEÆ SEMILUNARES, one upon either side, passing from the ninth costal cartilage to the pubic bone and following the outer border of the rectus abdominis muscle.

(c) THE LINEÆ TRANSVERSÆ, of which there are three, the upper being at the level of the tip of the ensiform cartilage, the middle at a level midway between the first and the navel, and the third at the level of the navel.

(d) In fat persons A DEEP TRANSVERSE SULCUS or furrow crosses the abdomen a short distance above the pubic arch and a second similar but less marked groove is sometimes seen about the level of the umbilicus. These grooves vary in depth according to the amount of fat in the belly wall and are deeper in the erect than in the recumbent posture.

The Imaginary or Conventional Lines.

(a) THE MESIAL LINE, passing through the tip of the ensiform cartilage, the umbilicus, and the symphysis pubis, and corresponding to the linea alba.

(b) THE PROLONGATION DOWNWARD of the midclavicular line, which passes through the eighth costal cartilage to the middle of Poupart's ligament upon each side.

(c) THE INFRACOSTAL LINE, passing around the body in the horizontal plane of the tenth costal cartilages.

(d) THE BI-ILIAC LINE, which corresponds to the plane of the most prominent part of the iliac crests.

These two lines (c) and (d) divide the abdominal surface into three zones: an upper or epigastric, a middle or umbilical, and a lower or hypogastric. The two vertical lines dropped from the middle of the clavicle to the middle of Poupart's ligament again divide each of those zones into three regions, as follows:

(a) AN EPIGASTRIC REGION OR UPPER CENTRAL REGION.—This overlies a portion of the right and left lobes of the liver and a large part of the anterior wall of the stomach, with the pylorus, the aorta, the cœliac axis, the semilunar ganglia, and at a greater depth the pancreas.

(b) A Right and Left Hypochondriac Region.—The right hypochondriac region overlies the right lobe of the liver and the gall-bladder, the duodenum, the hepatic flexure of the colon, and the upper part of the right kidney; the left the greater curvature of the stomach, the spleen, the tail of the pancreas, the splenic flexure of the colon, and the upper part of the left kidney.

(c) An Umbilical or Middle Central Region.—In this space lie the greater curvature of the stomach, the mesentery, the great omentum, coils of the small intestine, and the transverse colon.

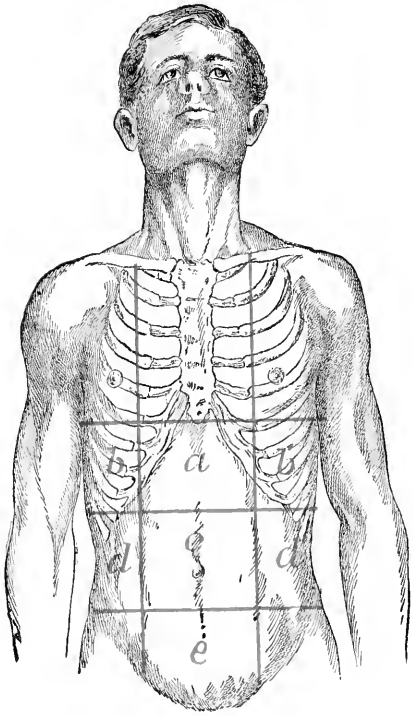


Fig. 15.—Regional divisions of the abdomen: *a*, epigastric or upper central region; *b,b'*, right and left hypochondrium; *c*, umbilical or middle central region; *d,d'*, right and left lumbar regions; *e*, hypogastric or middle lower region.

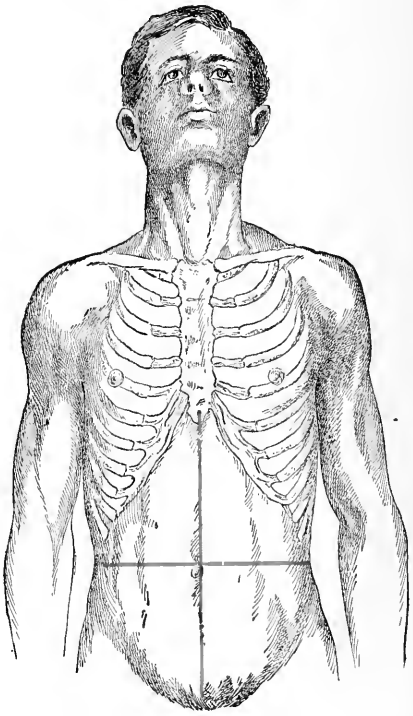


Fig. 16.—Quadrants of the abdomen.

(d) A Right and Left Lumbar Region.—The right contains the lower part of the right kidney, the ascending colon, and coils of small intestine; the left the lower part of the left kidney, descending colon, and small intestine.

(e) A Hypogastric, Suprapubic, or Middle Lower Region.—This space overlies coils of the small bowel, at its lower portion the fundus of the urinary bladder when distended, and the gravid womb.

(f) A Right and Left Iliac or Inguinal Region.—The right contains the cæcum and the base of the appendix or frequently the whole of it, the ileocæcal valve and the right ureter; the left the descending colon and left ureter.

The Quadrants of the Abdomen.

A simpler division of the surface of the abdomen into regions may be made by a vertical and a transverse line intersecting at the umbilicus. The four spaces thus defined are known respectively as the RIGHT and LEFT UPPER and LOWER QUADRANTS.

The Visceral Regions.

Certain important viscera give their names to the surface areas corresponding to the situation in which they are normally found. Thus we speak of:—

(a) THE PRECORDIAL AREA; THE PRECORDIA.—That part of the chest wall which overlies the heart, including the areas of superficial and deep dulness and increasing in extent in cardiac dilatation and hypertrophy.

(b) THE REGION OF THE APEX.—A more circumscribed space immediately above and around the normal apex and shifting as the apex shifts in enlargement and displacement of the heart.

(c) THE GASTRIC AREA, which corresponds to the normal situation of the stomach. The limits of this region are not strictly defined, since the organ varies in size when empty or distended with food or gas, and has some degree of mobility.

(d) THE HEPATIC AREA.—The lower border of this region is usually sharply defined both in normal and pathological conditions. Its upper border rounds away from the chest wall from which the upper surface of the liver is separated by the edge of the lung, and its left border is obscured by the tympany of the stomach and colon.

(e) THE REGION OF THE GALL-BLADDER.—The notch for the gall-bladder lies in the under border of the liver, slightly internal to the ninth right costal cartilage and near the outer border of the right rectus muscles. The fundus of the organ when distended and enlarged occupies a considerable area on both sides of this point as well as below it.

(f) THE ILEOCÆCAL AREA.—The part of the abdominal surface lying in the right lower quadrant of the abdomen and the seat of the local manifestations in appendicitis. Here lies the spot of focal tenderness described as McBurney's point.

(g) THE SPLENIC AREA.—The region which occupies the left hypochondrium extending towards the infra-axillary region. An enlarged spleen frequently transcends the normal borders of the splenic area, and a dislocated spleen occupies an entirely different position, in such a manner that the normal dulness in the splenic area is replaced by tympany.

(h) THE SIGMOID AREA.—The left inguinal region and the parts bordering upon it toward the median line, which are so designated because new growths and other pathological conditions involving the sigmoid flexure of the colon give rise to tumors or other clinical manifestations in this portion of the abdomen. It corresponds with the left lower quadrant.

(i) The Pelvic Area.—The designation sometimes employed to describe the suprapubic area because it is the region of the abdomen in which enlargements and new growths of the pelvic viscera are frequently manifest.

The extent of the various regions of this group is neither constant nor well defined. Their borders are often shifting and overlapping. Nevertheless they serve a useful purpose in the diagnosis of diseases of the abdominal organs.

Large accumulations of fat in the belly wall or within the peritoneal cavity, pregnancy, meteorism, dropsy and ascites, visceral displacements and enlargements, new growths and extra- and intraperitoneal cysts and abscesses distend the abdomen, modify its contour, and disarrange, often to an extreme degree, the relations between the above-described areas and the internal organs.

The foregoing anatomical and conventional lines and areas enable us definitely to fix the position of clinical phenomena for purposes of description and record.

The signs or symptoms of a lesion may be referred to a given region, as episternal pulsation, infraclavicular dulness, or precordial pain. More exactly the location of a given phenomenon may be indicated by the rib or interspace in which it is found and the distance from the midsternal line or its relation to one of the other vertical lines described, as, for example, the signs of a small cavity in the second interspace, a measured distance to the right—or left—of the median line; a presystolic thrill in the fifth interspace, to the left of the left parasternal line; an undulatory impulse in the fourth, fifth, and sixth interspaces, extending to a point midway between the left midclavicular line and the line of the anterior axillary fold.

A tumor or painful spot in the abdomen may be located in one of the nine regions described as the epigastric, right iliac, hypogastric, and so on, or in one of the quadrants of the abdomen.

If greater accuracy is desired, the position of a lesion, physical sign, or tender spot may be stated to be a measured distance to the right or left, as the case may be, of the middle line at the level of the umbilicus, or a measured distance above or below the level of the umbilicus. Or, again, the anterior superior spine of the ilium may be taken as the point of departure for similar measurements.

In the back the spinous processes may be taken as points of departure for the measurements. Thus a lesion may be a measured distance from the middle line on a level with the eighth dorsal spine or over a numbered interspace or rib.

The unit of measurement may be the centimetre, or the inch, if preferred, or the finger's breadth which equals about 2 centimetres or $\frac{5}{4}$ inch, or the hand's breadth, which varies from about 9 to 11 centimetres or $3\frac{1}{2}$ to $4\frac{1}{2}$ inches.

It is customary to indicate the extent of a lesion or the size of a tumor by less accurate but significant anatomical measurements; thus we say of a splenic tumor that it extends to the crest of the ilium or to the symphysis pubis or beyond the median line, or of a distended bladder or enlarged uterus that it reaches halfway from the pubis to the umbilicus or to the level of that anatomical landmark.

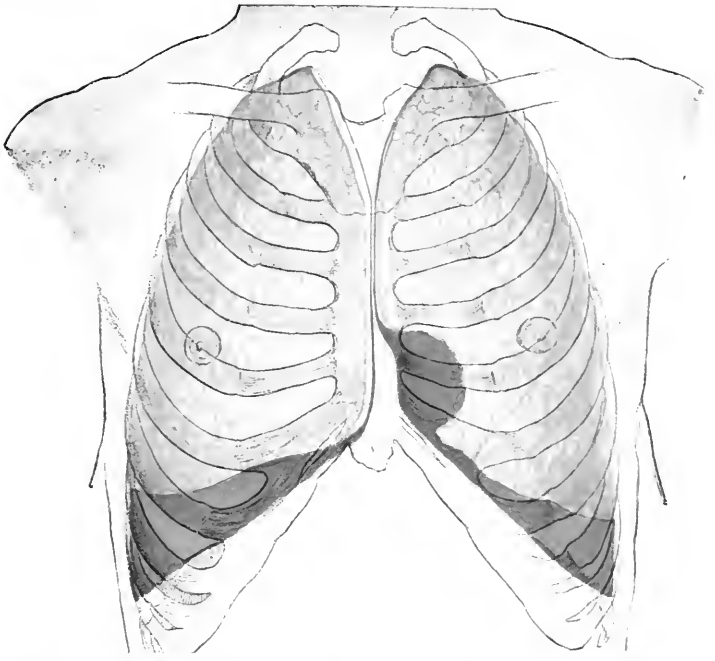


Fig. 17.—Semidiagrammatic reconstruction, showing relations of pleural sacs (blue) and lungs (red) to thoracic wall; anterior aspect.

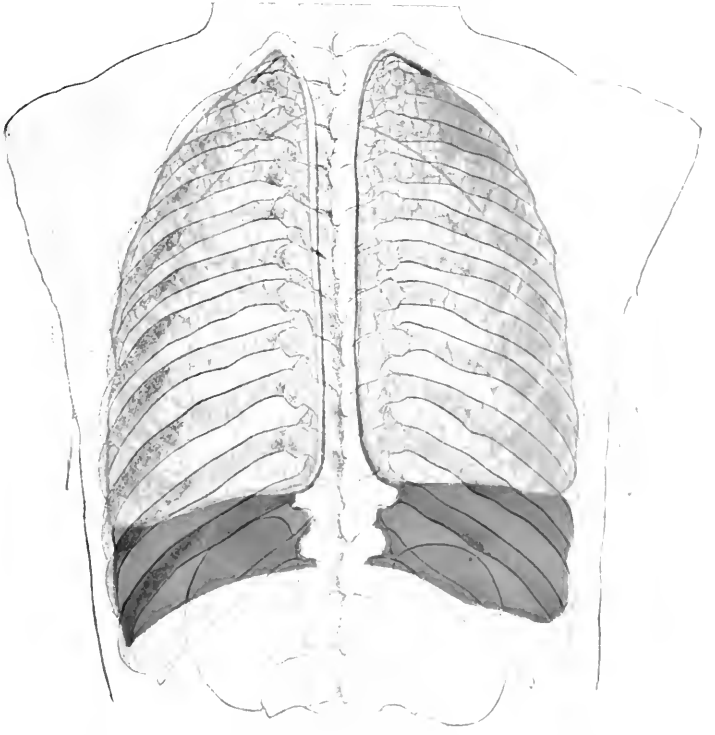


Fig. 18.—Semidiagrammatic reconstruction, showing relations of pleural sacs (blue) and lungs (red) to body-wall; posterior aspect.

THE TOPOGRAPHICAL ANATOMY OF THE THORACIC ORGANS.

The Thymus Gland and its Remnants.

This temporary organ attains its maximum development about the end of the second year. It then undergoes a gradual involution process until it is reduced to a mere vestige. When fully developed it appears as a narrow elongated body lying in the anterior mediastinal space immediately behind the manubrium sterni and extending into the episternal region of the neck. Its size varies according to the degree of development. At birth it is about 6 centimetres in length, 2.5 centimetres in width, and .75 centimetre in thickness. The thymus is occasionally persistent and may then undergo hypertrophy. In this case and when enlarged as the result of tuberculous, syphilitic, or cancerous disease, or hemorrhagic or purulent infiltration, pressure symptoms, namely, paroxysmal dyspnœa—so-called thymic asthma—persistent dyspnœa, spasm of the glottis, or venous hyperæmia and local œdema arise.

The Trachea or Windpipe.

This tubular organ extends in the median line from the larynx to a point opposite the third dorsal vertebra, where it is crossed in front by the arch of the aorta, and there or immediately below this level it bifurcates into the right and left bronchi. Its length is variable, being in the adult about 9 to 11 centimetres, its width from 2 to 2.5 centimetres. It is both wider and longer in the male than in the female. The trachea is movable and may be displaced as well as compressed by an aneurism or a new growth. Its posterior membranous part is in relation with the œsophagus behind, and the recurrent laryngeal nerves ascend in the groove between these two organs. The manubrium sterni overlies the trachea, which traverses the posterior mediastinum.

The Primary Bronchi.

The right and left bronchi arise at the bifurcation of the trachea and diverge to the corresponding lung upon each side, which they respectively enter at the root to form by successive subdivisions the ramifications of the bronchial tree. The right bronchus—the wider and shorter of the two—passes obliquely downwards and outwards to the lung at the level of the fourth dorsal vertebra, and behind the aorta; the left, smaller in diameter but much greater in length, runs obliquely downwards and outwards below the arch of the aorta to the root of the left lung, into which it passes at the level of the body of the fifth dorsal vertebra. The length of the right bronchus is about 2.5, that of the left nearly 5 centimetres.

Irregular stenosis of the trachea or a main bronchus, from an aneurismal or neoplastic tumor or from a tenacious and adherent exudate, causes tracheal stridor and the accumulation of an abundant liquid exudate, as in some forms of bronchitis, and the pulmonary œdema that precedes death gives rise to coarse tracheal râles.

Elasticity of the Tracheobronchial Structures.—That these organs have a high degree of pliability, analogous to that of the vesicular structure of the lung, is shown by the manner in which they accommodate themselves to the displacing and distorting pressure of effusions, aneurism, and new growths of various kinds without great impairment of their function. That they possess equally remarkable capacity of elongation and contraction has been recently demonstrated by X-ray examination and the bronchoscope of Chevalier Jackson.

The Œsophagus: Gullet.

This tubular organ extends from the pharynx at the lower border of the fifth cervical vertebra—the level of the cricoid cartilage—along the anterior surface of the borders of the vertebræ, to pass through the diaphragm about the level of the ninth dorsal vertebra and end in the cardiac orifice of the stomach. Its length is about 23 centimetres. In the thorax it lies posterior to the lower part of the trachea, the upper part of the left bronchus, and the posterior surface of the pericardium. The œsophagus may be the seat of simple or syphilitic cicatricial stricture; stenosis from cancerous growth involving its wall or pressing upon it from without or from the external pressure of an aneurism. Spasmodic stricture occurs in neurotic and hysterical persons, and the impaction of a foreign body, as an artificial denture, a large piece of meat, or a bone, may cause mechanical obstruction, an accident that occasionally occurs among the insane. It is sometimes the seat of a diverticulum. The œsophagus is accessible to examination by the sound, the œsophagoscope, and X-rays. The time occupied in swallowing and the nature of the accompanying sounds may be studied by auscultation.

The Lungs and Pleuræ.

The lungs occupy the greater part of the cavity of the chest, enclosing between their concave inner surfaces the heart and great vessels. Each lung is attached to the inner wall of the thorax in the region of the bodies of the fourth and fifth dorsal vertebræ by a comparatively small pedicle called the root, and a narrow membranous fold continued downwards from it. Elsewhere the surface of the lung is free and covered by a serous membrane, the pleura, which is also reflected upon the inner wall of the chest. The root of each lung is composed of the respective main bronchus together with large blood-vessels, lymphatic vessels, chains of lymphatic glands, held together by connective tissue and enclosed in the pleura.

THE PLEURÆ.

Each pleura is a closed serous sac, lining the lateral cavity of the thorax to which it belongs, enclosing the lung and its root and forming by the aid of its fellow of the opposite side the mediastinum. That part of the pleura which encloses and covers the lung and its root is called the visceral or pulmonary pleura; that which is reflected upon the ribs and intercostal spaces, covers the upper convex surface of the diaphragm, and passes to the sides of the pericardium, thus forming the mediastinum,

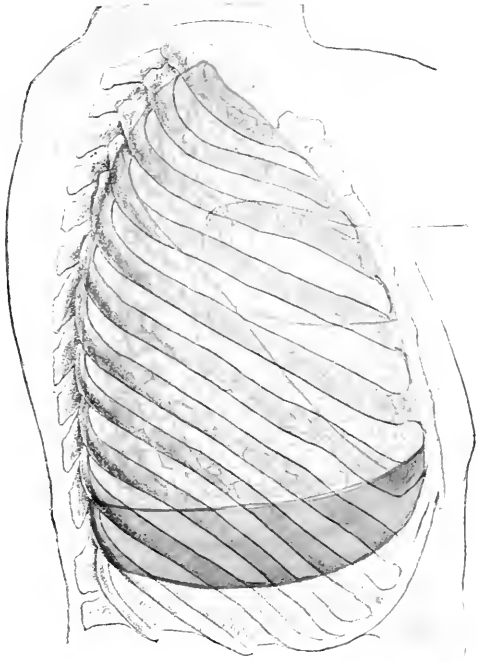


Fig 19.—Semidiagrammatic reconstruction, showing relations of right pleural sac (blue) and lung (red) to thoracic wall; lateral aspect.

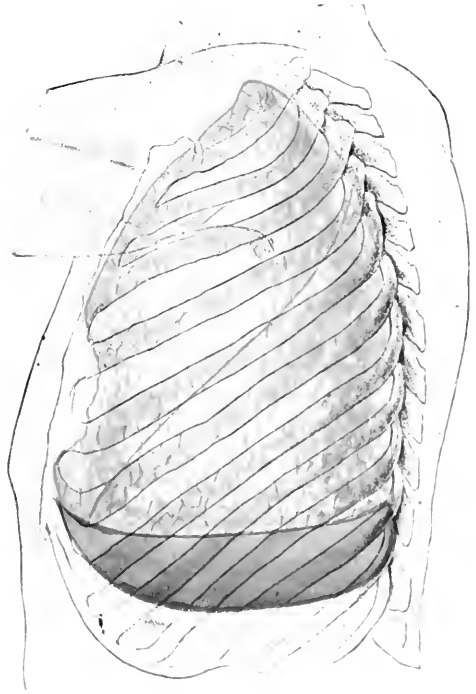


Fig. 20.—Semidiagrammatic reconstruction, showing relations of left pleural sac (blue) and lung (red) to thoracic wall; lateral aspect.

is called the parietal pleura, or—as to its different parts—the costal, diaphragmatic, and mediastinal pleura; and these two parts—namely, the visceral and the parietal pleura—are continuous with each other at the root of the lung.

The upper part of the pleura on each side passes upward beyond the clavicle into the neck, and contains the apex of the lung, which reaches from 2.5 to 4 centimetres above the margin of the first rib, usually a little higher upon one side than upon the other, but not constantly higher upon the right side as is often stated. Beneath the sternum the pleural sacs of the two sides come nearly or quite into contact in the upper part, but in the lower part the right pleura passes to or even beyond the middle line and the left pleura recedes from it to a variable distance beyond the sternal border. At the base of the chest the pleuræ do not reach to the attachments of the diaphragm, but they are reflected from the inner wall of the chest to the rising vault of the diaphragm in such a manner that, on quiet respiration or on full expiration, the parietal and visceral pleuræ are not in apposition, but the costal and diaphragmatic surfaces of the parietal pleura are opposed. The higher position of the right diaphragmatic vault, due to the high position of the right lobe of the liver, renders the right pleura somewhat shorter than the left, while the smaller portion of the heart upon the right side of the median line renders the right pleura somewhat wider than the left.

THE LUNGS.

Each lung is cone-shaped—with its blunt apex extending into the root of the neck, its anterior surface flattened, its lateral and posterior convex surfaces strongly convex, and its inner and inferior surfaces concave. The contour resulting from this conformation gives rise to sharp, well-defined anterior margins, the horizontal sections of which are acutely angular, and to a similar, sharply angular, circumferential border at the base, which fits into the corresponding re-entrant angle between the thoracic wall and the diaphragm—a fact of no little importance in physical diagnosis. Each lung is divided by a long, deep fissure, beginning about the level of the spine of the scapula and proceeding obliquely downward and outward to the sixth rib in the midaxillary line, into an upper and a lower lobe. The right lung is further divided by a second, shorter fissure, which passes inward either straight or in an upward or downward direction through the anterior margin, thus forming a third or middle lobe. Upon the inner anterior border of the left lobe is situated a deep notch into which the heart, enveloped in its pericardium, is received, and at the inferior part of this border of the lung is situated a tongue-like projection which passes in front of the apex of the heart—lingula.

The lungs completely fill the chest, and the surfaces of the visceral and parietal pleuræ are accurately in contact except along the anterior and inferior margins of the lungs. In these situations the sharp wedge-like borders of the lung advance between the reflected layers of the parietal pleura during inspiration and recede during expiration, as above stated.

The Mediastinum.

This space lies between the layers of an anteroposterior septum formed by the inner or mesial portions of the right and left pleuræ which pass upon the surface of the pericardium from the anterior and posterior walls of the chest to the root of the lung upon either side. It is subdivided into an anterior, middle and posterior mediastinum.

The anterior is narrow and of little depth, lying directly behind the inner surface of the sternum. At its upper part it contains the atrophied thymus. Behind the gladiolus the right and left pleuræ are in contact, and the anterior mediastinum consists merely of the connective-tissue layer by which they are joined. Lower down, while still shallow, it is widened, by the departure of the left pleura from the midsternal line, into a triangular space which lies between the anterior portion of the right ventricle and the wall of the thorax—the area of superficial cardiac dulness.

The middle mediastinum is the large space between the mesial layers of the two pleuræ which contains the pericardium and its contents.

The posterior mediastinum lies in front of the vertebral bodies and contains the trachea, the œsophagus, the thoracic duct, the descending aorta, the azygos vein, lymphatic vessels and the pneumogastric nerves.

THE PERICARDIUM.

This membranous sac, which occupies the middle mediastinum and contains the heart and the roots of the great blood-vessels, is conical in shape, its base resting upon the diaphragm and its apex extending upwards upon the walls of the blood-vessels as far as their first subdivisions. It consists of two layers, an external fibrous layer, which is attached below to the central tendon of the diaphragm, and above to the surface of the large blood-vessels which it embraces, and an inner serous layer, which lines the fibrous sac in which the heart is contained and is reflected upon the surface of that viscus in such a manner as to form a parietal and a visceral portion. The latter is sometimes described as the epicardium. The fibrous pericardium is furthermore firmly attached to the structures by which it is surrounded, namely, the sternum in front, the mediastinal pleuræ laterally, and the trachea, œsophagus, and main bronchi behind.

The Heart and Great Vessels.

THE HEART.

This central organ of the circulation is situated in the cavity of the thorax in the middle mediastinum. It lies unattached within the pericardium except by the great vessels which spring from its cavities at the base, and it rests upon the convexity of the diaphragm. Its base is directed upward, backward, and toward the right, and extends from the level of the fourth to that of the eighth dorsal vertebra, while its apex points downward, forward, and toward the left, coming into relation with the chest wall in the fifth intercostal space a little to the left of the parasternal line. It projects farther to the left of the median line than to the right in the average ratio of nearly 2 to 1.

Orthodiagraphic measurements have shown that the average oblique diameter of the heart from the true apex to the angle at the upper right border of the auricle and the great vessels is between 13 and 14 centimetres; the horizontal distance from the midsternal line to the most distant point of the border of the heart on the right, 3.5 to 4.5 centimetres; to the most distant point on the left, 7.5 to 8.5 centimetres.

The Relation of the Heart to the Anterior Wall of the Chest.—In general the normal heart in the adult may be said to extend from the level of the second intercostal space on the right side to the fifth interspace on the left. Investigations conducted to ascertain the exact relations of the viscus to the chest wall by thrusting long needles through it immediately after death, by means of sections of frozen bodies, and by the X-rays have not yielded constant nor concurrent results. The discrepancies are doubtless due to differences existing naturally among individuals and to variable conditions, in themselves equally incapable of exact determination: for example, the position of the diaphragm, the amount of residual air in the lungs, the quantity of gas in the stomach and intestines, and the volume of blood in the chambers of the heart at the time of examination. For clinical purposes it is possible to be overexact in variable matters of this kind.

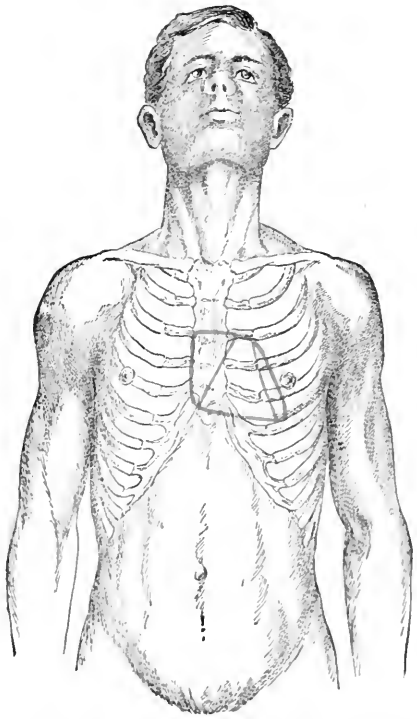


FIG. 21.—Outline of heart and lines indicating the auriculoventricular groove and the anterior interventricular groove.

The greater part of the anterior surface of the heart is not directly in relation with the inner chest wall, but separated from it by the wedge-like anterior borders of the lungs. The superior border of the heart closely corresponds to a transverse line drawn about the level of the upper edges of the third costal cartilages and extending from a point two centimetres from the right border of the sternum to the third left costochondral articulation. This line constitutes the clinical base of the heart and subdivides the precordia into the cardiac area and the area of the great vessels.

The inferior border is indicated by a line drawn from a point on the upper border of the sixth rib, directly below the outer limit of the impulse, obliquely upward and to the right, across the base of the ensiform cartilage, and terminating at the middle of the fifth right interspace near its junction with the sternum.

The right border nearly coincides with a line drawn from the point at which the superior border terminates on the right, convex to the right, to the middle of the fifth interspace as above, namely, about 2 centimetres to the right of the right sternal border.

The left border is marked by a line joining the apex and the articulation of the third left rib with its cartilage.

A line joining the third left chondrosternal articulation and the seventh right chondrosternal articulation corresponds fairly well with the line of the auriculoventricular septum.

A line joining the apex and the third left costochondral articulation corresponds closely with the interventricular septum.

The greater part of the anterior surface of the heart is formed by the right ventricle and constitutes a triangle included between the above lines and the inferior border of the heart. The apex of this triangle is occupied by the conus arteriosus and the tip of the left auricular appendix.

The upper third of the right auricle lies behind the sternum, while its two lower thirds extend to the right of the sternal edge and are bounded by the curved right border of the heart.

The left auricle is deeply seated and is completely covered by the body of the heart and the left lung.

The left ventricle is likewise deeply seated and wholly retired from the surface of the chest with the exception of a narrow longitudinal strip which forms the left border of the heart and presents anteriorly, and of which the lower end constitutes the true or anatomical apex of the heart, and is separated from the chest-wall by the lingula, the clinical apex to which the impulse is due being the apex of the right ventricle.

That portion of the anterior surface of the heart which, uncovered by the borders of the lungs, comes into relation with the wall of the chest, constitutes the area of superficial cardiac dulness and may be more or less accurately defined by percussion; that which recedes by its rounded surfaces from the chest wall and is covered by a rapidly thickening volume of lung tissue is described as forming the area of deep cardiac dulness and cannot be defined with the nicety which some assume by the ordinary methods of physical diagnosis, though the shadow of its borders may be seen expanding and contracting with the revolutions of the heart upon X-ray examination.

THE GREAT VESSELS.

The ascending arm of the arch of the aorta arises at the base of the left ventricle of the heart behind the pulmonary artery. Its course is at first upward and to the right and slightly forward as it passes behind the sternum. At the level of the second right costal or aortic cartilage, the vessel passes upward, backward, and to the left, forming the transverse portion of the arch, then backward and downward to form the descending arm of the arch which terminates in the descending portion of the thoracic aorta.

The pulmonary artery passes a little more than a centimetre beyond the left border of the sternum in a line about the level of the middle of the left third interspace upward to the second costal cartilage, behind which it divides into its right and left main branches.

The descending vena cava extends from the second interspace on the right side of the sternum to the base of the heart, which it enters at the level of the middle of the third interspace. Its course is slightly curved, the convexity being toward the right.

These vessels are situated at varying depths behind the manubrium sterni and in an area extending beyond the right and left sternal borders. This region is sometimes designated the area of the great vessels.

The Relation of the Valves of the Heart to One Another and to the Surface of the Chest.—The lines of attachment of the bases of the mitral and tricuspid valves correspond to the auriculoventricular sulcus. The semilunar cusps of the aortic and pulmonary valve systems are situated respectively at the origin of each of those vessels from the ventricles. The four sets of valves lie in close proximity to one another and to some extent overlap. The pulmonary is most superficial; the mitral most deeply situated; the aortic centrally placed and in parts of its extent covered by the pulmonary; and the tricuspid lowest in position.

Their relations to the surface of the chest are as follows:

The pulmonary valve lies horizontally immediately to the left of the sternal border at the level of the upper edges of the third left costal cartilage.

The aortic valve is at a level slightly lower than the pulmonary and situated behind the sternum at the level of the third left intercostal space and to the left of the median line. It is nearly horizontally placed.

The mitral valve—left auriculoventricular — lies on an oblique line from above downward to the right behind the sternum extending from the level of the left third intercostal space to the level of the lower border of the left fourth costal cartilage.

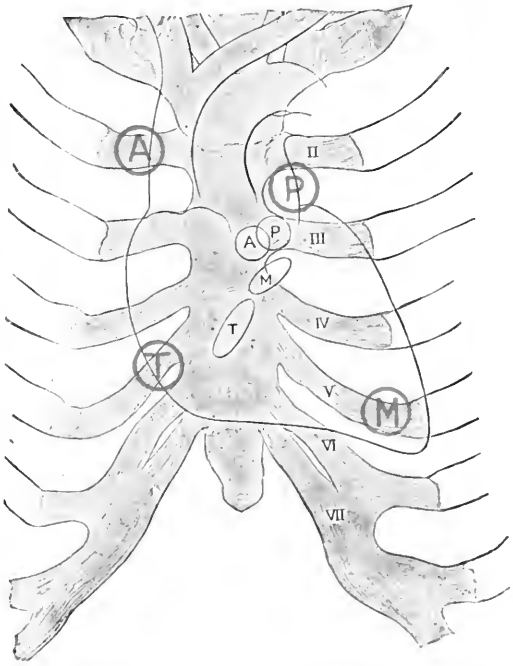


FIG. 22.—Position of heart and valves in relation to anterior thoracic wall. A, aortic valve; P, valve of pulmonary aorta; T, tricuspid valve; M, mitral valve; and puncta maxima indicated by red circles.

The tricuspid valve—right auriculoventricular—lies still more obliquely behind the sternum opposite the fourth intercostal space on a line drawn through a point in the midsternal line on the level of the third interspace to the sixth chondrosternal articulation.

These four valve systems are so close to one another that the sounds produced by each cannot be studied by auscultation directly over the seat of the valve, but at that point in the precordia at which the blood stream at the moment directly affecting the particular valve mechanism approaches the surface of the chest most closely.

Puncta Maxima.—These areas, of which there are four, corresponding to the separate valve systems, are:

1. **The pulmonary area**—at the inner end of the second left intercostal space.

2. **The aortic area**—at the second right costal cartilage.
3. **The mitral area**—at and just above the position of the apex-beat.
4. **The tricuspid area**—at the right border of the lower end of the sternum.

THE TOPOGRAPHICAL ANATOMY OF THE ABDOMINAL VISCERA.

The Stomach.

The stomach is that dilated portion of the alimentary canal which lies between the cardiac end of the œsophagus and the pyloric end of the duodenum. It is irregularly gourd-shaped, the larger left end being called the fundus or splenic extremity; the smaller right end the pyloric extremity. The orifice by which the œsophagus enters is called the cardia or cardiac orifice, that passing to the duodenum the pylorus. The former is immediately below the central part of the diaphragm and lies between the greater and lesser curvatures. The latter lies lower down, more toward the anterior abdominal wall, and to the right. The shorter inner curvature of the gourd is known as the lesser, the longer outer curvature is the greater curvature of the stomach. This hollow viscus lies chiefly in the epigastric and left hypochondriac regions, the greater part of its extent being, when distended, in about the proportion of 1 to 5, to the left of the median line. During physiological rest the healthy stomach contains only a little mucus and a small accumulation of air or gas which occupies its fundus, and forms a narrow wrinkled pouch, the long diameter of which is oblique from the cardia downward and to the right and approaches much more nearly to the vertical than to the transverse axis of the body. Its superior border is fixed at the cardia at the point at which the œsophagus pierces the diaphragm and is attached to the overlying liver and diaphragm by the gastrohepatic omentum and the gastrophrenic ligament. The gastrocolic omentum is attached to the lower, the gastrosplenic omentum to the left border. The anterior surface is in relation with the diaphragm and under surface of the liver above and the wall of the abdomen lower down; the posterior surface is in relation with the great vessels and pancreas above and the transverse mesocolon lower down. Both these surfaces are free, smooth, and invested with peritoneum. When the stomach is distended, it rotates upon its cardiopyloric axis in such a manner that the anterior surface tends to look upward and the posterior surface downward. The dimensions of the stomach vary according to the degree of distention caused by food, fluid, or gas. When moderately filled, its longest diameter is about 25 centimetres, its diameter between the greater and lesser curvature from 9.5 to 12 centimetres, and the diameter between its anterior and posterior walls about 9 centimetres. When much distended, a normal stomach may reach to the level of the umbilicus.

The cardia is situated in a direct line posterior to the left seventh chondrosternal articulation at a distance of about 10 to 12 centimetres from the anterior abdominal wall. The pylorus, which has considerable freedom of motion, lies about the level of the tip of the ensiform cartilage and near the outer border of the right rectus muscle. It is in relation with

the concave surface of the liver and may extend to the neck of the gall-bladder. When the stomach is distended the pylorus assumes a position further to the right and lower in the abdomen. The fundus rises into the vault of the diaphragm to the level of the fifth interspace in the midaxillary line and is higher than the cardia, just as the lateral vault of the diaphragm is higher than its central aponeurosis. Its upper part lies behind the anterior diaphragmatic border of the left lung and the tips of the seventh, eighth, and ninth left ribs and their cartilages. The convex curve of Traube's semilunar space in this region corresponds with the curvature of the fundus of the stomach.

The Intestines.

A. **The small intestine** begins at the pylorus and terminates at the ileocæcal valve, at which point it joins the large bowel. It has an average length in the adult of about six metres. Its convolutions occupy the middle parts of the abdomen and are surrounded by the large intestine. They are attached to the back wall of the abdominal cavity by the mesentery. The small intestine is divided into (1) an upper portion, or duodenum, about 25 to 30 centimetres in length, into which in its middle third the common bile duct and pancreatic duct discharge their contents; (2) a middle portion, or jejunum; and (3) a lower portion, or ileum. In the last are situated Peyer's patches. The duodenum is the widest and least movable of the three portions of the intestines. The coils of the jejunum and ileum are freely movable within the abdomen and among themselves and bear no constant relation to the regions of the surface.

B. **The large intestine** extends from the termination of the small intestine at the ileocæcal valve to the anus. Its average length is between 1.5 and 2 metres. Its diameter varies at different parts and ranges from 3.5 to 6 centimetres. There is a pouch-like dilatation of the rectum immediately above its lower end. It is divided into three parts.

(1) **The Cæcum; Intestinum Cæcum; Caput Cæcum Coli.**—The shortest and widest part of the large intestine. It measures in length and width each about 6 centimetres. As a rule, there is no mesocæcum, and this part of the intestine is attached to the fascia covering the right iliacus muscle. The cæcum is situated in the right iliac fossa and is comparatively fixed. Its position determines that of the ileocæcal valve which lies between 6 and 7 centimetres mesial to the right anterior superior spinous process.

(2) **The appendix vermiformis** arises from the inner and posterior aspect of the cæcum near the ileocæcal valve. It lies in the right iliac region and its base is opposite McBurney's point. Its dimensions are extremely variable, its width being that of a large quill and its length from 6.5 to 9 centimetres. From its comparatively fixed base, the appendix, being free, may extend in any direction. As a rule it lies downward or inward. It may, however, extend backward, in which case the symptoms of appendicitis may suggest renal colic; or upward, and, if inflamed, suggest gall-bladder disease.

(3) **The Colon.**—This part of the large intestine constitutes its greatest length. It occupies the peripheral parts of the abdominal cavity, and, owing to the lack of a mesocolon in its ascending and a portion of its descending course, maintains a comparatively fixed position. In some

instances there is a short mesocolon in these portions. It is divided, according to its course and direction, into four parts, namely, an ascending, a transverse, a descending portion, and the rectum.

(a) The ascending colon, commencing at the cæcum, passes upward in a vertical direction to the under surface of the liver near the gall-bladder, where it turns forward and sharply to the left, forming the hepatic flexure. It is as a rule fixed in its whole course and overlaid by some coils of the ileum. It is contained in the right lumbar and hypochondriac regions.

(b) The transverse colon passes across the umbilical region from the right to the left hypochondrium. It is deeply situated at its right and left extremities, but in its intermediate course it bends forward and approaches the anterior wall of the abdomen—arch of the colon. It rises slightly at its left extremity to pass behind the costal margin in relation with the fundus of the stomach and turns abruptly downward to form the splenic flexure.

(c) The descending colon is continuous with the transverse colon at the splenic flexure. It descends nearly directly downward through the left hypochondrium and lumbar region to the left iliac region, where it curves inward and then downward to form the sigmoid flexure. The descending colon is covered only in front and at its sides by peritoneum, but the sigmoid flexure has a distinct mesocolon and is freely movable. The latter lies well toward the front of the cavity of the abdomen in the left iliac region.

(d) The rectum, notwithstanding its name, is not straight in man, but curved from its beginning at the brim of the pelvis in front of the left sacro-iliac articulation obliquely downward from left to right to the middle line of the sacrum, then forward in the hollow of the sacrum to the level of the prostate in the male or the vagina in the female, where it again turns and proceeds downward and obliquely backward to the anus. This part of the large intestine lies entirely within the pelvis, but is accessible to examination by the finger, the rectal bougie, and the proctoscope.

The Liver.

The liver is the largest gland in the body and occupies a great space in the abdominal cavity. It measures from 22 to 24 centimetres in its transverse, about 15 centimetres in its maximum anteroposterior, and 14 to 16 centimetres in its maximum vertical diameter. It is bulky and rounded in its right extremity; narrow and wedge-shaped toward the left; convex and smooth upon its upper surface; concave, uneven, traversed by various fissures, and showing the gall-bladder and extrahepatic bile passages upon its lower surface. The rounded, thick posterior part is the most fixed; the thin, sharp anterior margin the most movable part of the organ.

The liver occupies the right hypochondriac and extends across the epigastrium into the left hypochondriac region. It is closely adapted to the vault of the diaphragm and is in relation with the anterior wall of the abdomen on the right side as far down as the margin of the ribs. The right lobe reaches higher than the left—a fact in accord with the shorter vertical diameter of the right thorax as compared with the left. At its highest point the convex upper surface of the right lobe of the liver corresponds to the fourth intercostal space in the midclavicular line. The upper

boundary gradually declines to the base of the ensiform cartilage in the direction toward the left and continues on the right and to the back almost upon the same level, crossing the midaxillary line at the level of the seventh intercostal space and the line of the angle of the scapula about the level of the ninth rib. Owing to the dome-like shape of the upper surface of the right lobe of the liver and the concavity of the base of the lung into which it is adapted, the diaphragm being interposed, there is a considerable difference in the level of the actual upper border of the organ and that of the portion which lies in contact with the wall of the thorax. The latter

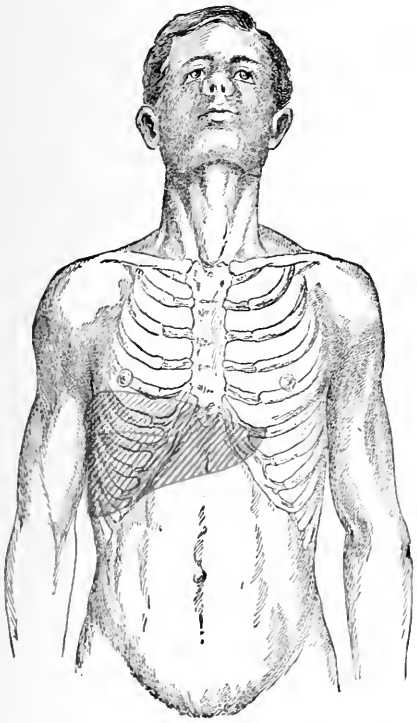


FIG. 23.—Areas of deep and superficial hepatic dulness.

FIG. 24.—Areas of deep and superficial hepatic dulness.

in the midclavicular line corresponds with the sixth rib; in the midaxillary line with the eighth rib, and posteriorly with the tenth rib. Upon percussion that portion of the liver which lies in relation with the wall of the chest yields well-marked dulness; that which is covered by the interposed border of the lung modified dulness. The former is spoken of as the area of superficial hepatic dulness, the latter as the area of deep hepatic dulness, and these two areas together constitute the area of hepatic dulness.

The lower anterior margin corresponds in the midclavicular line with the margin of the ribs; in the median line it lies slightly above a horizontal line midway between the base of the ensiform cartilage and the umbilicus; about the left parasternal line at the lower border of the sixth rib; in the right midaxillary line at the tenth interspace; and at the spine about the level of the eleventh intercostal space.

The interlobar notch lies nearly in the median line. The thin edge of the left lobe reaches closely to the midclavicular line. To the right of the right midclavicular line the lower border corresponds approximately to the costal margin. In aged persons the liver occupies a slightly higher level; in children it is large in proportion to the size of the body and extends higher, displacing the apex beat of the heart to a point behind the fifth rib or in the fourth interspace, and causing the lower border to fall below the line above indicated by 1 or 2 centimetres.

The Gall-Bladder and Extrahepatic Bile Passages.

THE GALL-BLADDER.

This membranous sac is situated in a fossa in the base of the liver. It is pear-shaped, measuring in its long diameter from 7 to 10 centimetres and in its greatest transverse diameter about 4 centimetres. It lies obliquely, with its fundus, which projects beyond the anterior margin of the gland, looking downward, forward, and to the right. There is often a slight notch in the margin of the liver at this point, which corresponds to the outer border of the right rectus muscle at the level of the inner edge of the ninth costal cartilage.

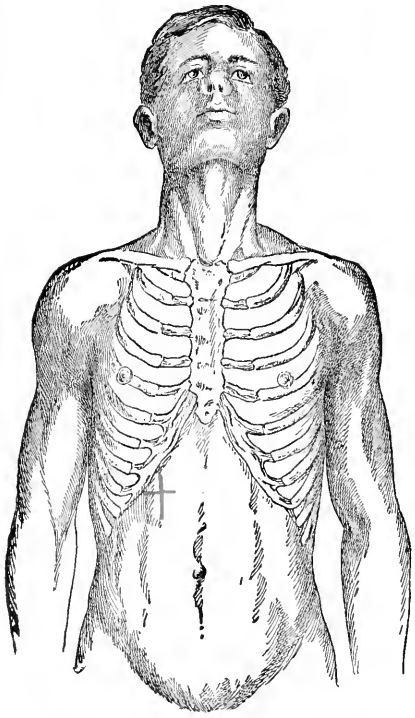


Fig. 25.—Position of fundus of gall-bladder.

THE EXTRAHEPATIC BILE PASSAGES.

The Cystic Duct.—The neck of the gall-bladder, which grows gradually narrower, forms a double curve like the letter S, and then becoming much constricted it turns abruptly downward to form the cystic duct, which runs downward and to the left and unites with the hepatic duct to form the common duct.

The Hepatic Duct.—This duct is formed by the union of a right and a left branch, which issue from the transverse fissure and unite at an obtuse angle. Its diameter is 3 or 4 millimetres and its length about 4 centimetres. It unites with the cystic duct to form the common duct.

The Common Bile Duct; Ductus Communis Choledochus.—This is the largest of the bile passages, being 5 or 6 millimetres in width and 6 centimetres or more in length. It runs downward and backward to the inner and posterior wall of the duodenum, where, uniting with the pancreatic duct to form a dilatation, known as the ampulla of Vater, it penetrates the wall of the duodenum very obliquely in the course of its middle third.

Pathological conditions involving the ducts, such as cholangitis and gall-stone disease, do not directly give rise to physical signs, but they cause serious symptoms and, indirectly, marked physical signs, and a knowledge of the position and size of these ducts and their relations to each other is of prime importance in the diagnosis of the diseases to which they are liable.

The weight of the liver and its direct relationship with the diaphragm render it to a high degree subject to the influence of gravity in different postures of the body, as, for example, the erect position as compared with the dorsal decubitus, and to the influence of the respiratory movements. Due allowance for these changes in the position of the organ is to be made in its physical examination.

The Pancreas.

This elongated, flattened gland is situated deeply in the abdominal cavity directly behind the stomach and at the level of the first lumbar vertebra. The larger right extremity is called the head and is embraced by the curvature of the duodenum. Its smaller left extremity, the tail, is situated in a slightly higher level than the head and reaches to the spleen, with which it is in contact. This organ varies considerably in size, being between 15 and 20 centimetres in length, about 4 centimetres in average breadth, and about 2.5 centimetres in thickness. It extends across the epigastric region and into the right and left hypochondrium. Its principal duct traverses the entire length of the gland and in association with the common bile duct enters the duodenum by an oblique passage through its wall. Its great depth in the body renders it as a rule inaccessible to direct physical examination. The close relations of the head of the pancreas with the portal vein, the inferior vena cava, and the ductus communis choledochus are of clinical importance, since malignant or other disease attended by enlargement of that part of the gland constitutes a not infrequent cause of œdema, ascites, or persistent jaundice.

Fig. 26.—Position of spleen.

The Spleen.

This soft, vascular organ is situated in the left hypochondrium, opposite the ninth, tenth, and eleventh ribs, and in the posterolateral portion of the upper part of the abdominal cavity. It undergoes considerable variation in size in health and may be enormously enlarged in disease. It is irregularly oval in shape, its upper and posterior borders being rounded and thick, its lower and anterior borders sharp and the latter indented by two or more notches. Its convex outer surface is in relation with the inner surface of the left side of the diaphragm. Its concave inner surface presents a vertical fissure called the hilus, and is in relation at its posterior portion with the

suprarenal capsule and the upper part of the left kidney, and at its anterior portion with the stomach, the splenic flexure of the colon, and coils of the small intestine. Its average long diameter under normal conditions is between 8 and 10 centimetres and it cannot be felt upon palpation. Supernumerary spleens are not uncommon.

The Kidneys.

The right and left kidneys are deeply seated in the lumbar region in the back part of the cavity of the abdomen and behind the peritoneum, opposite the last dorsal and the first, second, and sometimes the third lumbar vertebræ. The position of the right kidney is slightly lower than that of the left. Each kidney is about 9 centimetres long, 6.5 centimetres in width, and 3 centimetres in thickness, the left being usually longer and thinner than the right. Their oblong, rounded concavo-convex shape is characteristic. The convexity of each is directed outward and backward; the concavity inward and slightly forward. Near the middle of the concave surface is a longitudinal fissure or hilus at which the vessels and nerves enter or emerge and the ureter arises. This excretory duct expands within the hilus into the pelvis of the kidney, from which arise three or sometimes two funnel-shaped spaces which subdivide into a number of smaller tubes called calices or infundibula, similarly funnel-shaped but into which the papillæ of the kidney project. The kidneys are supported by the vessels and the perirenal fat. The right kidney is in relation with the duodenum and colon in front and the liver above; the left with the spleen above and colon anteriorly. Both lie against the corresponding pillar of the diaphragm, the anterior layer of the lumbar fascia, and the psoas muscle. The deep situation of the kidneys and the thick layers of muscles against which they rest, embedded in a layer of fat behind, render them under normal circumstances inaccessible to the ordinary methods of physical examination. When they are displaced or enlarged they present characteristic physical signs. The suprarenal bodies are also beyond the reach of the usual procedures of physical diagnosis. The ureters descend from the hilus of each kidney to enter the bladder at its base. When dilated—hydronephrosis—they form characteristic abdominal tumors.

The Bladder.

When empty this organ lies below the symphysis pubis; when distended it gives rise to a globular area of dulness in the hypogastrium. In some neglected cases of urethral stricture or enlarged prostate an overdistended bladder forms a large fluctuating tumor, reaching as high as the umbilicus and inclining somewhat more to one side of the median line than to the other.

III.

THE EXAMINATION OF THE PATIENT AND CASE-TAKING.

Case-Taking.

An accurate knowledge of the facts in the case constitutes the first requisite to a diagnosis. Those relating to the medical life of the patient and his illness up to the time of his coming under observation are known as the HISTORY OF THE CASE, or the ANAMNESIS; those relating to his immediate circumstances, alike subjective or objective, are described under the heading PRESENT CONDITION, or STATUS PRÆSENS.

The examination to ascertain the necessary facts should be conducted in an orderly and systematic manner. Time is thus saved, a general survey of the clinical phenomena made, and those of chief importance brought into contrast and proper relation with those of subordinate value. Data not otherwise obvious are brought to light and the chances of oversight minimized. Vague and pointless inquiries are omitted. The interrogation is precise and explicit. Above all, leading questions are to be avoided. Running comments in the presence of the patient produce an especially unfavorable effect. Tact and patience are necessary. An examination thus conducted has a favorable influence upon the patient, especially in chronic and difficult cases, and always inspires confidence. The investigation should not be unduly extended or minute. The examination of an experienced and thoroughly trained clinician stands in striking contrast to the vague and unsystematic questions of the beginner. On the other hand, the inquiry may be too concise and brief. The former method has been spoken of as the extensive, the latter as the intensive. The middle course is the best.

There are two principal modes of case-taking, the synthetic and the analytic.

THE SYNTHETIC METHOD.

In the synthetic, sometimes spoken of as the historical method, the inquiry begins with the history of the patient, rather than with his present condition. His place of birth, age, social state, occupation, previous diseases, habits, hereditary and constitutional tendencies are first ascertained, then follows an investigation into the beginning and progress of the present illness. All this constitutes the anamnesis. The status præsens is then considered. The condition of the several physiological systems, the digestive, the circulatory, the respiratory, the genito-urinary, the nervous, and so on, being carefully inquired into in regular order. Finally, the symptoms and signs referable to the organs or structures especially affected are carefully studied. The next step in the process is the diagnosis, upon which the prognosis, treatment and general management of the case depend. Case-taking by this method follows the natural order. It is scientific and

useful in obscure cases. The chief objections to it are the time it consumes and the fact that in the progress of the inquiry unnecessary attention must be given to facts which are found later to have little or no bearing upon the patient's present condition.

THE ANALYTICAL METHOD.

In the analytical method the order of procedure is reversed. The principal symptoms are taken as the point of departure for the investigation. The organ or region to which these symptoms are referred is examined by the proper diagnostic measures. The general condition of the patient, his facies, the state of nutrition of his body, his posture, his movements, are carefully observed; meanwhile he is questioned as to the duration and progress of the present illness and an inquiry is made into such facts in his previous history and antecedents as may bear upon the case. The clinical study is then extended, the condition of the other organs investigated, the history of the case more systematically reviewed, an opinion formed as to whether the malady is general or local and a diagnosis reached. This is the plan commonly pursued in ordinary professional work where the data are sufficient for a diagnosis by the direct method, and is available in all cases except those where the symptoms are obscure and ill defined.

QUESTIONS.

Great care is necessary in formulating questions. It is not sufficient to ask the patient if the present illness began with a chill and be content with an affirmative answer. Many patients regard the transient shivering which so often marks the onset of an acute febrile disease as a chill, whereas it is a very different matter from the prolonged and intense rigor that attends the onset of pneumonia or the malarial paroxysm. The physician must be on his guard also in regard to statements made by patients or their friends concerning their previous illnesses. Very often such diagnoses are popular rather than professional, and questions must be so framed as to determine their accuracy. Accounts of influenza, malaria, catarrh of the stomach, rheumatism, and the like cannot be accepted without close investigation into the symptoms, course and duration of the illnesses referred to. The "stomach cough" and "malaria" of the consumptive are familiar to all practitioners. In the matter of hereditary and family tendencies to disease the examination must be conducted with great care. It is no uncommon thing for patients, even those who are well informed and intelligent, to deny the existence of malignant disease, chronic nephritis, a tendency to tuberculosis, and the like, when careful inquiry or the independent statements of their friends render the occurrence of these diseases in the family in the highest degree probable. A patient will affirm that no case of consumption has ever occurred in his family, and upon cautious questioning admit that his father or mother or other near relative suffered from chronic cough, abundant expectoration, blood-spitting, and progressive emaciation. An epileptic will deny the occurrence of nervous diseases, and subsequently admit that near relations have presented the symptoms

of hysteria or neurasthenia or been insane. Patients very often withhold in the presence of a nurse or other attendant important facts that they willingly communicate to the physician alone.

RECORDS.

Records should be kept in private as well as in hospital and dispensary practice. How full these should be will depend upon the physician's estimate of the importance of the individual case. Their preparation demands close attention, concise statements, and accuracy. They constitute a permanent store of professional experience for future reference and study. They are of great value in the review of the history of patients previously seen, as an aid in comparing one's personal observations with those of the profession at large, in the preparation of articles for publication, and not infrequently as bearing upon medico-legal cases. They should be preserved in accordance with a uniform plan in books prepared for the purpose, or preferably upon cards of convenient dimensions arranged in cabinets, in the same manner as the index catalogues used in libraries. Uniformity is important. It prevents the oversight of significant facts and facilitates the comparison of cases. The following scheme is suggestive; it may be modified in accordance with individual views:

SCHEME FOR CASE RECORDS.

Case record number.....Diagnosis.....Revise.....Result.....
Admitted.....Discharged.....(In hospital patients).
Date of examination.....
Name.....Age.....Sex.....Race.....Place of birth.....Present abode.....Former occupation.....Present occupation.....Social state..... Married, single, widowed.

Anamnesis.

1. Family History: Hereditary tendencies; health of parents, brothers and sisters; deaths in family—cause, age.

2. Personal History: (a) Diseases of childhood; (b) menstruation; (c) pregnancies, miscarriages, date of last confinement; (d) previous illnesses or injuries; (e) habits—regularity of meals, kind of food, method of eating; bowels; sleep; habitual or occasional physical or mental overexertion; tobacco; alcohol; narcotics.

3. Present Illness: (a) Date of onset; supposed exciting cause; exposure to contagion; prodromes; initial symptoms; course of the attack; previous treatment. (b) Antecedent derangements of health not amounting to positive disease, appetite, pain, cough, disturbances of sleep, headache, etc.

Status Presens.

A. General Appearance: Expression, height and weight, musculature, bony structure, panniculus adiposus; posture in bed; movements, gait and station out of bed; temperature; pulse; respiration; color and condition of the skin; perspiration; œdema; eruptions; psychical condition; sensations and complaints; delirium; convulsions, stupor; coma.

B. Particular Phenomena: Symptoms and signs relating to special structures, organs and functions.

1. The Digestive Apparatus: Inspection of the mouth, tongue and gums; tonsils and pharynx; palpation of the abdomen, its form and contour, visible peristalsis, tenderness upon pressure, resistance, tumors; percussion and palpation of the stomach and intestines, liver, gall-bladder, spleen; inspection of vomited matters and fæces.

2. The Circulatory Apparatus: Inspection and palpation of the cardiac area; visible and palpable pulsation; thrill; precordial prominence; position of the apex; percussion and auscultation of the heart; the pulse-frequency, rhythm, fulness, tension; condition

of walls of arteries; venous pulsation; capillary pulse; liver pulsation; auscultation of the arteries and veins; arterial pressure, maximal and minimal; examination of the blood, etc.

3. The Respiratory Apparatus: Nose, mouth, and larynx; cough and expectoration; chest and lungs—character of the respiration, dyspnœa, stridor, Cheyne-Stokes respiration; contour of the thorax; local, lateral or bilateral retraction or expansion; respiratory excursus; fremitus; local and general physical signs obtained by percussion, auscultation, and mensuration; the cyrtometer.

4. The Genito-Urinary Apparatus: Palpation of the kidneys and bladder; percussion of the bladder; retention of urine; suppression; frequency of micturition; pain; quantity of urine; total amount for twenty-four hours; disturbance at night; chemical and microscopic examination of the urine; sexual organs; the prostate.

5. The Nervous System: Intelligence; mental state; subjective sensations; sleep, gait, station, reflexes, tremor, convulsions, spastic conditions, paralysis; aphasia and other disorders of speech; derangements of sensation: the organs of special sense.

6. The Osseous System—Bones and Joints: General and local changes in the skeleton; cranium, spine, thorax, pelvis, long bones, extremities; striking deformities; the joints; size and shape, color, pain, degree of impairment of function, fixation, disintegration.

7. The Tegumentary System: Itching, burning, tension, pain, inflammatory phenomena; presence and character of eruptions, macular, papular, vesicular, pustular; uniformity; polymorphism; hypertrophy and atrophy; cicatrices; pigmentary changes; animal and vegetable parasites; subcutaneous structures; enlargement or atrophy of thyroid body; lymph nodes; constitutional disturbances.

Diagnosis; Prognosis: Treatment; Subsequent observations.

The results of special clinical and laboratory examinations are to be incorporated under the appropriate headings. Among these are rhinoscopic and laryngoscopic, ophthalmoscopic and otoscopic examinations; hæmatologic investigations; the chemical and microscopic examination of the gastric contents, vomited material, and the stools; of expectorated matters; bacteriologic examinations of the blood, sputum, secretions, exudates, etc., by the methods of staining, culture, and inoculation; examination of the rectum by the finger, the speculum, and by inflation; cystoscopy; special examination of the genital organs in both sexes, examination of the fluids obtained by exploratory puncture, and examination by the X-rays, etc.

In febrile cases temperature charts should be preserved with the records, and superficial deformities, as swelling or retraction, as well as changes in the viscera revealed by the various methods of diagnosis, may be indicated upon outline clinical diagrams and incorporated in the notes. Changes of contour, glandular enlargements and topographical lesions, such as local consolidations and cavity formation in the lungs, cardiac dilatation or hypertrophy, pleural and pericardial effusions and the resulting displacement of adjacent viscera, enlargement of the liver and spleen, dilatation of the stomach and displacement of the abdominal organs may in this manner be more or less accurately delineated. The location of tumors, circumscribed exudates, and other foci of infection may also be indicated, and in the case of the nervous system the extent and distribution of areas of disturbance of sensation and other phenomena.

Some further explanation of the bearing of the facts noted in the anamnesis upon the mental processes by which a diagnosis is reached may be of service to the student.

Age.—The age is important. Each period of life has its peculiar susceptibility to morbid influences. In the new-born, congenital defects, the results of the accidents of parturition, diseases arising from faulty management of the cord, those directly transmitted from the mother, and those produced by improper diet and unhygienic surroundings are

common. In childhood, anatomical peculiarities of the growing organism and the sensitiveness of physiological processes to external influences give rise to special predispositions to disease. Thus, the ready proliferation of the lymph tissues explains the frequent occurrence of respiratory obstruction in the nasopharynx from adenoid hypertrophy, while the narrowness of the larynx accounts for the gravity of catarrhal and infective processes involving that organ, and the great vascularity and rapid overgrowth of the epithelium of the bronchi when irritated explain the peculiar liability of children to bronchitis and bronchopneumonia. In the instability

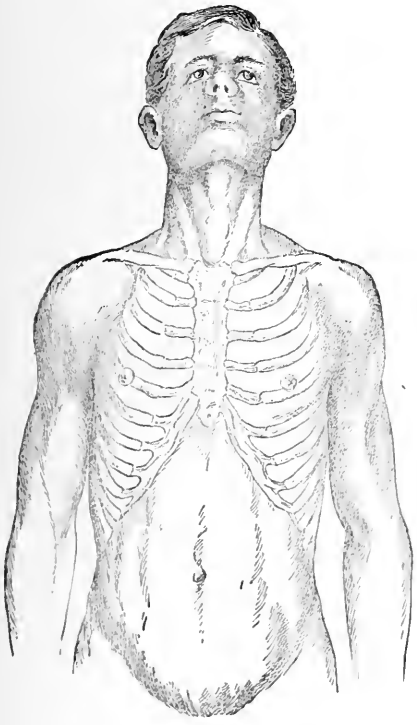


Fig. 27.—Clinical diagram.

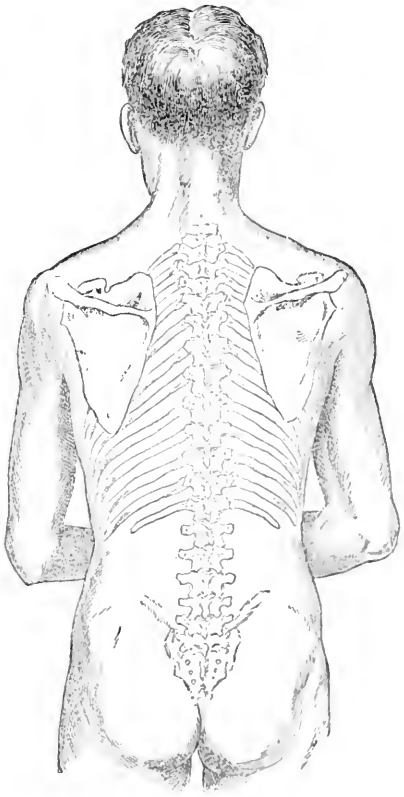


Fig. 28.—Clinical diagram.

of the nervous system in children we find a ready explanation of their liability to fever, its high range and rapid fluctuations, and to various reflex disturbances, and in the absence of acquired immunity, an explanation of the wide prevalence among them of the transmissible infections, as the exanthemata, which are spoken of as the diseases of childhood. In adolescence, hereditary predispositions begin to show themselves, as in the occurrence of tuberculosis and of epilepsy or other nervous affections. The late sequels of infantile diseases, as chronic valvular trouble following rheumatic endocarditis, or chronic nephritis subsequent to scarlatina, often now appear. Changes in the environment of the individual subject him to special pathogenic influences, and pleurisy with or without effusion,

pneumonia, and enteric fever are common. The middle period of life is especially prone to diseases that result from occupation, examples of which are lead intoxication, caisson disease, and scrivener's palsy, to those which result from the habitual use of narcotics, as gastric catarrh, hepatic cirrhosis, and alcoholic neuritis, to those resulting from the stress of life and anxiety, among which may be named cardiac hypertrophy, the neurasthenias and other nervous diseases and insanity. It is in this period that hereditary and acquired tendencies to sclerotic changes in the vessels and in the nervous system begin to develop and that diabetes and the paroxysms of gout commonly first show themselves. Later in life the indications of progressive degenerations become more marked. The wrinkled skin, the failing sight and hearing, the feeble heart, winter cough, and renal inadequacy are the indications of sclerotic and nutritive changes which are more apparent in the rigid, tortuous, or atheromatous superficial arteries. This is especially the period of apoplexy, chronic bronchitis, diabetes, cystitis from hypertrophied prostate, Parkinson's disease and the special infections, erysipelas and pneumonia, which are frequently terminal events. In general terms the evolution of life is the period of infections, the involution the period of degenerations; but in pathology age cannot be measured by years, and the significant saying that "a man is as old as his arteries" has become a modern medical aphorism.

Physiological Epochs.—The epochs of life are also marked by special liability to disease. Thus at the first dentition nutritional diseases and gastro-intestinal troubles are common; at puberty, chlorosis and hysteria; at the menopause, hysteria, obesity, and arthritis deformans. It is to be noted, however, that the maladies of these physiological epochs are not always the direct result of functional changes, but usually the outcome of previous morbid conditions or tendencies.

Sex.—Sex is likewise important. In early and advanced life the sexes are equally liable to disease. Women between the age of puberty and the menopause are exposed to the danger of many accidents and diseases peculiar to the anatomical and physiological development connected with the sexual life and child-bearing. Consideration of these matters properly belongs to gynæcology and midwifery. Sedentary living, the monotony of the household, and depressing moral influences also act as causes of disease in women. Hysteria, neurasthenia, and special forms of insanity occur. These peculiarities do not, however, carry with them an exemption from other pathogenic influences, and among the peasantry of those countries where the women largely engage in the same occupations as the men they are, in addition to their own peculiar disorders, equally liable to most of the maladies which affect the other sex and nearly to the same extent. In more enlightened districts and among the upper classes of society women escape many risks of disease to which men are exposed. In the male sex occupation, exposure, the strenuous life, and self-indulgence are common causes of disease, hence the more frequent occurrence of plumbism, farcy, pneumonia, chronic arthritis, gout, tabes, and alcoholism. As a consequence, arteriosclerosis and atheroma are more marked in men than in women at advanced age.

Race and Nationality.—These points demand consideration in the anamnesis. The peculiar liability of the Hebrew to diabetes and neurasthenia; of the negro and mulatto to tuberculosis, and the relative immunity of the former to malaria and yellow fever; the prevalence of beriberi among the oriental races, of leprosy in Scandinavia, the Sandwich Islands, and the West Indies; and the frightful ravages of tuberculosis, syphilis, and alcoholism among the Indians of North America are well-known facts.

Nativity. — The place of birth and residence frequently shed light upon an obscure case, as in ill-defined malaria, the malarial cachexia, goitre, cretinism, and leprosy. A knowledge of the district or locality of the patient's present residence, the situation of his home, its sanitary conditions and surroundings, the source of the water supply, and the disposition of the sewage may shed light upon the diagnosis.

Occupation.—The occupation of the patient demands careful investigation. The habitual over-use of certain muscles, and exposure to particular irritants or poisons or an atmosphere laden with minute mineral or metallic particles or chemicals, or to infections peculiar to certain crafts, cause definite diseases. Examples of such affections are writer's cramp, anthracosis or miner's consumption, chronic phosphorus poisoning among workmen engaged in the manufacture of matches, malignant pustule or wool-sorter's disease, and glanders. It is necessary to inquire carefully into former occupations as well as the present; thus chronic bronchitis with bronchiectasis may have had its origin in the inhalation of the dust caused by stone-cutting—an occupation long abandoned by the patient. In those occupied in professional or literary work functional derangements of the stomach, constipation, and insomnia are common. Even amusements may be the cause of disease, as in the golfer's back and the heart-strain of the athlete.

Heredity. — The family history has a very important bearing upon the diagnosis, especially in chronic diseases. It is difficult to frame a satisfactory definition for heredity, but we know that traits and lineaments are transmitted from parents to children through the generations, and we occasionally observe in a son who has his mother's features some trick of expression that makes his resemblance to his father for the moment almost startling. So too are transmitted from one generation to another tissue peculiarities and constitutional tendencies to disease. The inquiry into the family history must be, as has been pointed out in a previous paragraph, conducted with tact and caution. Blunt inquiries in regard to "consumption," "cancer," "Bright's disease," or "insanity" irritate the patient and usually elicit vague replies or absolute denials. A patient should be asked if his parents are living and in good health; if not in good health, the symptoms and duration of the illness; if dead, the cause of death and the age at which it occurred. He should be questioned as to the number of his brothers and sisters, their health, and the cause of any deaths that may have occurred among them. It is very important to learn whether or not deaths in the family have been the result of acute or chronic disease. The inquiry may be extended to the preceding generation and collateral branches of the family. Diseases, it is true, are conveyed by hereditary transmission, but their number is comparatively few. Hæmo-

philia is a striking example. Syphilis is very commonly thus transmitted. When the mother has contracted an acute infection, as measles or enteric fever, the child may be born during the period of incubation or with the symptoms of the disease already manifest. A number of nervous diseases are clearly hereditary. As examples may be mentioned progressive muscular atrophy, hereditary chorea, Friedreich's ataxia, and migraine. The definite symptoms may not show themselves for some years after birth, in some cases not until adult life. Much more commonly it is the predisposition that is transmitted. This is especially the case in tuberculosis. The peculiar exposure of the young infant to infection from a tuberculous mother and the length of time that the tuberculous lesions in many instances remain localized render it in the highest degree probable that the predisposition to tuberculosis rather than the disease itself is hereditary. This view is confirmed by the results of pathological and bacteriological investigations. The direct transmission of tuberculosis from the mother to the fœtus in the human being is of uncommon occurrence. The doctrine of the direct hereditary transmission of tuberculosis, so long entertained but now fortunately abandoned, was a stumbling block in the way of the recognition of the infectious character of this disease. That the predisposition rather than the disease is hereditary is also true of cancer. The occasional occurrence of chronic Bright's disease in nearly every member of a family in two or three generations, usually first showing itself in adolescence or early adult life, must be attributed to hereditary defects of the renal and vascular tissues, while faults of metabolism, the constitutional tendency to which is transmitted from father to son, bear a direct etiological relation to gout and its associated cardiovascular and renal changes. The development of forms of insanity in successive generations of a family, usually at the physiological epochs of life, often not until late middle age, must likewise be attributed to hereditary defects of nervous and mental organization.

A further peculiarity in regard to the hereditary transmission of disease is to be found in its diverse manifestations among various members of a family. The radical defect or susceptibility may find expression in pathological conditions which are allied but which have wholly different symptoms. Thus the tendency to deranged metabolism and arteriosclerosis may in one show itself in contracted kidney and hypertrophied heart; in another in disease of the aorta or angina pectoris; in a third in gout, renal calculus and gravel, or yet again in early cerebral hemorrhage or thrombosis. The neuropathic constitution may manifest itself in one member of a family in forms of neuralgia, neurasthenia, or hysteria; in another in the development of epilepsy, and in a third in the guise of hypochondriasis or insanity. The family susceptibility to certain infections may reveal itself in different individuals in recurrent attacks of tonsillitis or rheumatism, chorea or chronic valvular disease; or the susceptibility to tuberculosis, on the one hand in pulmonary consumption, on the other in tuberculosis of the bones and joints or glandular disease, or finally in the implication of the meninges, pleura, or peritoneum.

Immunity may be transmitted by inheritance as well as the predisposition to disease. There are families and individuals who possess a

remarkable natural immunity against the exanthemata. This is especially true in regard to scarlet fever. When we consider the wide prevalence of pulmonary tuberculosis and the diffusion of its cause in the centres of population and certain districts and houses, and the fact that so large a proportion of individuals and families constantly exposed to the inhalation of an atmosphere containing the tubercle bacilli escape the disease, the common existence of a natural immunity which is frequently transmitted by inheritance becomes evident. The predisposition to tuberculosis is far less general than that to scarlatina and measles. The occurrence of personal peculiarities and morbid tendencies in an individual which were not manifested in his parents but existed in their ancestors is known as atavism. In rare instances, and especially in cases of nervous disease and insanity, this condition is important in the anamnesis. Curious facts in regard to the duration of life are occasionally observed. There are families in which in successive generations few members survive the early middle period of life. In such instances death is very often due to an acute disease not always the same. On the other hand, all the members of certain families reach an advanced age, the exceptions being where death is due to accident or violence.

Medical History.—The personal history is essential to a diagnosis in the broad sense. A knowledge of the significant facts in the past life of the patient may clear up a doubtful case. The present disease may be a late sequel of some previous illness, as bronchitis or emphysema after whooping-cough, or an obscure manifestation of one of the exanthemata which the patient escaped in childhood, as scarlatina in the adult with fever of moderate intensity and an irregular patchy eruption, or it may be the expression of a peculiar constitutional susceptibility, as tonsillitis, rheumatic fever, or chorea, from which the patient has suffered on previous occasions. In this connection it is to be borne in mind that many of the acute infectious diseases, and especially the exanthemata, result in an acquired immunity which usually lasts throughout life, hence second attacks are exceedingly infrequent, while the immunity conferred by other infections, for example rheumatic fever, erysipelas, croupous pneumonia, and diphtheria, is incomplete and of limited duration, so that many individuals suffer from repeated attacks of these diseases. In acute febrile attacks and in the presence of epidemics careful inquiry as to exposure to the contagion must be made. The period of incubation and the occurrence of prodromal symptoms are to be taken into consideration. In women abnormal menstruation, the accidents and diseases of pregnancy, the occurrence of miscarriages, too frequent child-bearing and prolonged lactation may be the cause of serious impairment of health or of actual disease. These matters must be carefully inquired into. In exceptional cases, especially in aggravated and intractable functional nervous diseases, it becomes necessary to inquire more closely into the sexual life of the patient. The investigation must be conducted with great delicacy and discretion. The part played by vicious practices and excesses in the production of such diseases must be ascertained. It is necessary also to learn whether or not the patient has suffered from venereal infection, the date of its occurrence, the nature, character, and duration of the primary symptoms, the presence

or absence of secondary lesions, and the treatment. Gonorrhœa is not always merely a local affection. The frequency with which it is followed by stricture is well known, but the symptoms of the latter condition may first show themselves after the lapse of years. Local abscess formation, acute and chronic cystitis and pyelitis also occur. The immediate recognition of the specific nature of gonorrhœal ophthalmia, whether in the new-born or in the adult, is a matter of overwhelming importance. The diagnosis of many a case of disabling and stubborn arthritis is made clear by a knowledge of gonorrhœal infection. Nor is the fact to be overlooked that endocarditis, both in its benign and malignant forms, may be a secondary process. In women the history of primary gonorrhœa is very often obscure. Tubal disease and other pelvic inflammations, only to be relieved by the knife of the gynæcologist, are common results of the extension of the infection. A dissolute life on the part of the patient is presumptive evidence of the nature of the process. There is also gonorrhœa insontium; a virtuous wife may suffer. The protean manifestations of syphilis are to be borne in mind. The symmetrical arrangement and sequence of the early cutaneous lesions, their later polymorphism and irregular distribution, the buccal and anal mucous patches, the adenopathy, the obscurity of the visceral and nervous phenomena, their irregularity and chronicity, are all to be considered in the diagnosis of an obscure case. The presence of the specific organism—spirochæta pallida—is conclusive. Where syphilis is suspected in a family, we must inform ourselves as to whether or not a mother has aborted, especially in her early pregnancies, or has had later a series of abortions or still-born children, and as to snuffles and cutaneous eruptions, especially on the buttocks, in her new-born children, and corneal opacities, interstitial keratitis, Hutchinson's teeth, and arrested development or nervous diseases in those who have survived. Nor must the physician overlook the fact that many innocent persons contract syphilis. Not only the blameless wife but also the unsuspecting girl, from the kiss of her betrothed, may become the victim to this disease, while the methods of accidental inoculation are innumerable. Familiar examples are to be found in the chancre upon the hand of the surgeon or accoucheur, or upon the lip or tongue of the incautious borrower of a pipe from an infected friend. When matters of this kind concern members of a family, the physician cannot be too guarded in respect to the way in which his questions are framed or in his statements to a husband or wife. Suggestive questioning or injudicious statements may seriously aggravate existing troubles. If definite communications become necessary, his knowledge of the circumstances will enable him to decide whether it is best personally to assume the whole responsibility or to invite a colleague of high reputation to share it with him.

The history of a surgical operation and the conditions which led up to it, as well as its results, are important. The patient's present condition may be due to a recurrence of the original trouble, or, as in the case of an abdominal operation, to the development of adhesions or constricting bands.

Personal Habits.—The habits must be closely studied. Important information bearing upon the diagnosis may often be obtained by directing the patient while continuing his ordinary method of living to keep a

record of the hours at which his meals are taken, the kind and quantity of food and drink, the action of his bowels, the hours and character of sleep, and his various occupations and amusements, which may be submitted at a subsequent consultation. The causal relation of improper clothing to bronchopulmonary affections, of badly regulated work and sleep to neurasthenic conditions, of injudicious or irregular eating to gastro-intestinal troubles, of the abuse of alcohol to nervous diseases and cirrhosis of the liver, of excess in tobacco to irritable heart and amblyopia, will guide us in the inquiry. Late hours and dissipation, in fact all matters which enter into consideration from the stand-point of the moral hazard of the insurance companies, have a most important bearing upon diagnosis.

Present Illness.—The history of the present illness must be systematically investigated and its symptoms recorded in chronological order from the onset to the time of the patient's coming under observation. It is important to learn if possible the effect of treatment. The disappearance of a rash after mercurials or the subsidence of headache after continued large doses of the iodides constitutes presumptive evidence in favor of syphilis. The failure of quinine to prevent the recurrence of chills renders the diagnosis of malaria improbable, or of the proper administration of suitable preparations of iron in full doses to correct the pallor, breathlessness upon exertion, and headache of a highly anæmic young woman militates against the diagnosis of chlorosis. Much allowance must be made for the statements of patients both as regards the symptoms of the illness and their reports of previous treatment and the opinions of physicians whom they may have consulted. In many cases the unravelling of a diffuse and inconsequent story can only be accomplished by the exercise of skill and patience. On the other hand, the history communicated by intelligent persons is often curiously succinct and clear. Frequently by reason of the patient's mental condition no account of the illness can be obtained. In some cases it often happens that very little information can be gleaned from the bystanders. In hospital practice the admission of ambulance cases gravely ill, of whose previous condition nothing whatever can be learned, is a matter of daily occurrence.

Duration.—Of first importance is a knowledge of the duration of the illness, since it enables us at once to form an opinion as to whether the disease should be referred to one or the other of the two general groups of acute or chronic maladies. The fact is, however, not to be overlooked that acute symptoms may be the manifestation of an unsuspected chronic affection, as sudden loss of vision or convulsions in nephritis, angina pectoris in disease of the heart and aorta, or perforation phenomena and peritonitis in peptic ulcer of the stomach and duodenum. The mode of onset next demands our attention. In chronic cases we seek information as to whether the present illness developed insidiously or abruptly upon a condition of previous good health, or followed an acute illness, and whether its course has been gradual and progressive or interrupted by periods of improvement; in acute cases whether the attack developed insidiously, as in the case of enteric fever, or abruptly, as in influenza or typical croupous pneumonia, and whether or not prodromes occurred. It is next in order to ascertain the prominent symptoms of the disease, the region or organ

to which they have been referred, whether they have been continuous, intermittent, or paroxysmal, and any changes in the patient's appearance or condition, of which he may or may not be aware, that have attracted the attention of his friends. Finally, important information is often reached by due consideration of the views of the patient or others relating to the cause of his illness.

Status Præsens.—The investigation of the present condition of the patient must also be conducted in an orderly and systematic manner. The subjective sensations are carefully considered. No complaint of the patient, however trifling, is to be wholly disregarded. The objective symptoms must be studied with equal care. Every fact is to receive proper consideration. Due regard must be paid to the feelings of the patient. Abruptness and all appearance of haste or harshness are to be avoided. The interview must not seem too business-like. The clothing, whether in the consulting room or at the bedside, must be so arranged as to facilitate the examination. No physical exploration of the thoracic or abdominal organs can be made without proper access to the regions to be studied; mistakes from a disregard of this rule are of daily occurrence. In diseases of the heart, lungs, or great vessels it is necessary to inspect the uncovered chest; palpation must also be performed upon the bare surface; percussion and auscultation upon the bare skin or more conveniently in most cases through a towel or the single layer of a smooth under-vest. In order that the influence of gravity upon the abdominal viscera may be learned or to study the station and gait, the patient must rise from bed. If there are symptoms referable to the spine, the clothing must be removed and the patient examined in the erect, sitting, or recumbent posture, in the last instance not in bed but upon the firm, smooth surface of a suitable table; the effect of various movements is studied and the condition of the muscles and joints. Accurate measurements of parts, preferably in centimetres, are essential where there is a departure from normal standards or asymmetry. We measure and note the circumference of the head in hydrocephalus, the chest on quiet breathing, on full held inspiration and on forced expiration, its lateral circumferences for comparison, its contour by means of the cyrtometer, and we may measure diameters of the head and chest by means of calipers. It frequently, especially in the case of ascites and tumors, is desirable to take the circumference of the abdomen The muscles in relaxation and contraction are studied by the hand and one side is compared with the other. Where necessary the circumference of the limbs is measured at the same level upon the two sides. Where symptoms relating to the brain or spinal cord dominate the clinical picture, the examination must be made with especial attention to the details bearing upon the localization of the lesions. Specimens of the urine must be obtained for examination as a matter of routine in all cases. The diagnosis of obscure conditions, the symptoms of which are referred to the nervous system, digestive organs, or general condition of the patient, frequently depends upon the result. The discovery of chronic disease of the kidneys or the presence of sugar in the urine as the outcome of investigations made upon application for life insurance is a matter of very common occurrence in middle-aged men who regard themselves as in excellent health.

In general the examination should be methodically conducted in accordance with the foregoing scheme, prominence being given in the record of the case to the symptom-complex which bears directly upon the diagnosis.

Abbreviations.—Time and space may be saved in case-taking by the use of abbreviations. Thus:

f & m l & w—father and mother living and well.

b 3; 2 *d in infancy;* 1 *l & w*—three brothers; two dead in infancy; one living and well.

s 2; 1 *d at* 7 *sc fever;* 1 *at* 10 *acute nephritis.*

w & s till 18 *then ent fever; l crural phlebitis; elas stk still*—well and strong till 18, then enteric fever; followed by left crural phlebitis; still wears elastic stocking.

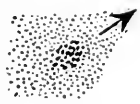
Epigast pain p c; occas v; blood 12 *mos & 1 mo ago*—Epigastric pain after food; occasional vomiting; hæmatamesis one year and again one month ago.

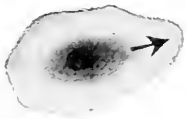
D r u a; def expn; br-vesic resp; crep râles—Dulness right side upper lobe, anteriorly; deficient expansion; bronchovesicular respiration; crepitant râles.

Tend r l q; circ D; 3d d of attack; n & v; T. 101°—Tenderness in right lower quadrant; circumscribed dulness; third day of attack; nausea and vomiting.

Many similar abbreviations, at once familiar to the writer and intelligible to any trained clinician, will suggest themselves.

Murmurs may be shown on the clinical diagrams (Figs. 27 and 28) by stippling or washes, the point of maximum intensity being most deeply colored and the direction of propagation shown by an arrow:



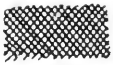


or more simply by a many-pointed star to indicate the point of maximum intensity and an arrow the direction; thus

These signs should be drawn in a different color from that of the ground plan, red if the latter is black, or *vice versa.*

Dulness may be indicated by cross hatching; its degree by closeness of the mesh; thus

Relative dulness.



Marked dulness

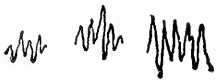
Flatness by solid color; thus

Râles by dots, their size and abundance corresponding to the physical signs; thus

Crepitant. Subcrepitant. Small mucous. Large mucous.

Cavities by irregularly outlined spaces; thus

Friction sounds by zigzags, the extent and coarseness of which indicate the distribution and intensity of the rub; thus



The Clinical History.—The result of the foregoing systematically conducted examination of the patient is known as the clinical history. It consists of four parts and is comprised under the following corresponding subheadings:

1. Anamnesis.—The account given by the patient or his friends of the history of his case up to the time of the examination.

2. Status Præsens.—A record of the results of the objective examination by clinical and laboratory methods.

3. Catamnesis (Barker).—The history of the patient's illness from the time of the examination, comprising the facts in the course of the disease and the details of treatment.

4. Epicrisis.—A methodical, critical judgment or review of the case with the discussion of questions arising from its consideration, especially as to the result of treatment or surgical operation or the post-mortem findings in case of death.

PART II.

OF THE METHODS AND THEIR IMMEDIATE RESULTS.

I.

MEDICAL THERMOMETRY.

THE art of taking and recording the temperature of the body is called medical thermometry. The instruments used are known as clinical thermometers. They are marked off in degrees upon the glass, and each degree is subdivided into fifths, so that the readings may conveniently be recorded in fractions of the decimal system. The thermometers commonly used in the United States and Great Britain are marked in degrees of *Fahrenheit's* scale; those used in Europe are graduated according to the *Centigrade* scale. The scale of *Réaumur* is rapidly going out of use, but is still employed in some parts of Europe. On the scale of Fahrenheit the distance through which the mercury rises from zero to the boiling-point of water is divided into two hundred and twelve degrees, of which the thirty-second marks the melting-point of ice. Between the melting-point of ice and the boiling-point of water there are one hundred and eighty degrees ($32° + 180° = 212°$ F.). The melting-point of ice is taken as zero in the Centigrade scale and in that of Réaumur, but in the Centigrade the boiling-point of water is at one hundred (100° C.), while in Réaumur's it is at eighty (80° R.). The relation of the three scales to each other is, therefore,—

$$\frac{\text{F.}}{9} \qquad \frac{\text{C.}}{5} \qquad \frac{\text{R.}}{4}$$

To convert recordings of the Fahrenheit scale into Centigrade degrees,—

Subtract 32, multiply by 5, and divide by 9; thus: $98.6 - 32 = 66.6 \times 5 = 333.0 \div 9 = 37$. That is, 98.6° F. = 37° C.

To convert Centigrade degrees into Fahrenheit degrees,—

Multiply by 9, divide by 5, and add 32; thus $37 \times 9 = 333 \div 5 = 66.6 + 32 = 98.6$. That is, 37° C. = 98.6° F.

The Centigrade scale is more convenient than that of Fahrenheit, and many physicians in this country prefer to use it. The following table of approximate equivalents may prove of use:

96.0° F. = 35.5° C.	101.3° F. = 38.5° C.	106.7° F. = 41.5° C.
96.8° F. = 36.0° C.	102.0° F. = 38.9° C.	107.0° F. = 41.6° C.
97.8° F. = 36.0° C.	102.2° F. = 39.0° C.	107.6° F. = 42.0° C.
98.0° F. = 36.6° C.	103.0° F. = 39.4° C.	108.0° F. = 42.2° C.
98.6° F. = 37.0° C.	103.1° F. = 39.5° C.	108.5° F. = 42.5° C.
99.0° F. = 37.2° C.	104.0° F. = 40.0° C.	109.0° F. = 42.8° C.
99.5° F. = 37.5° C.	104.9° F. = 40.5° C.	109.4° F. = 43.0° C.
100.0° F. = 37.8° C.	105.0° F. = 40.5° C.	110.0° F. = 43.3° C.
100.4° F. = 38.0° C.	105.8° F. = 41.0° C.	111.2° F. = 44.0° C.
101.0° F. = 38.3° C.	106.0° F. = 41.1° C.	

Seasoning.—As thermometers after a time give readings that are slightly too high, in consequence of the gradual contraction of the glass, it is necessary at long intervals carefully to compare them with a standard instrument. This is done at the public observatories, to which they may be sent. This contraction of the glass is called "seasoning," and goes on very slowly. After two or three years it practically comes to an end, and the thermometer is then seasoned.

Description of Thermometers.—Clinical thermometers as at present made are of the kind known as maximum, or self-registering; that is, a small portion of the mercury is separated from the main bulk of it, or separates itself from it as it contracts, by reason of a device in the twist of the tube, in such a way that it remains in position in the tube when the temperature falls, until shaken down, and thus indicates the highest temperature reached during the observation. The separated portion of the mercury is known as the "index." The reading is taken from the upper end of the index, which is then shaken down by a quick motion of the wrist, such as is made in cracking a whip, the thermometer being held by its upper end. Before taking the temperature the index should be below 95°. The best clinical thermometers are now made with a curved surface, which, acting as a lens, magnifies the width of the mercury; and with a flattened back, which lessens the danger of breakage from rolling. Aseptic thermometers have an outer glass tube encasing the engraved scale so that the external surface is perfectly smooth.

Technic.—The object being to measure the internal temperature, the thermometer must be placed in such a position that the tissues of the body completely surround its bulb. The positions available are the armpit, or axilla, the mouth, the vagina, and the rectum. The fold of the groin, when the thigh is bent up or flexed over the abdomen, is in infants also occasionally used.

The axilla is frequently selected. If very moist, it should be dried with a towel before the instrument is introduced; or, if dry and harsh, it must be bathed with warm water and then dried. There is no difference in the temperature of the two armpits under ordinary circumstances. The bulb of the instrument must be placed deeply in the hollow and the arm brought well across the chest. Care must be taken that no fold of clothing interfere with the contact of the instrument with the skin. Some thermometers are more sensitive than others; that is, they act more quickly. The mercury rises rapidly at first, then more slowly. Thick thermometers require five minutes to record the maximum temperature, but the best instruments now made reach the highest point in about two minutes. In the rectum or vagina less time is required.

When the temperature is taken in the mouth the bulb must be placed under the tongue and the lips closed about the stem, the patient breathing through his nose. It is an excellent plan to dip the instrument in water and wipe it with a clean napkin in the presence of the patient both before and after using it in the mouth. It is not safe to take the temperature in the mouth either in young children or in conditions of delirium. When the patient is in an insensible state, or when doubts arise as to the correctness of an axillary observation, the rectum or the vagina may be used for apply-

ing the thermometer, and with self-registering instruments this plan involves no exposure of the person. In European countries the common custom is to take the temperature in the rectum. In restless children care must be taken to prevent the instruments being broken, and in all cases to prevent a short thermometer from slipping entirely into the bowel, from which it might be difficult to extract it. The temperature may be rapidly taken in unmanageable children by means of an old-fashioned thermometer which is not self-registering, by cautiously warming it until the mercury reaches a very high point, say 108°, and then quickly placing it in the armpit. The mercury falls rapidly to the temperature of the patient's body and then stops.

Frequency. — It is desirable to take the temperature at least twice daily, the best times being between seven and eight in the morning and about eight in the evening. The observations must be repeated at the same hours each day. In cases characterized by great or sudden variations of temperature, by very high temperature, or when the influence of treatment upon the fever is being closely watched, observations must be made at shorter intervals of time, and it may become necessary to take the temperature as often as every hour.

Abnormal Temperatures.

The temperature in disease may range below or above the normal. Sudden falls of temperature in fever are very significant; just as are abrupt rises from the temperature of health. The following terms are used to indicate the general condition of the patient in abnormal ranges of temperature:

	F.	C.
Below the Normal.		
a. Temperature of collapse	Below 96.5°	35.8°
b. Subnormal temperature	96.5°— 98°	35.8°—36.7°
c. Normal temperature	98° — 99.5°	36.7°—37.5°
Above the Normal.		
d. Subfebrile temperature	99.5°—100.5°	37.5°—38 1°
e. Moderate febrile temperature	100.5°—102° A.M.	38.1°—38.9°
(Mild pyrexia)	102.2°—103° P.M.	39° —39.5°
f. High febrile temperature	102° —104° A.M.	38.9°—40°
(Severe pyrexia)	104° —105.8° P.M.	40° —41°
g. Intense febrile temperature (Hyperpyrexia)	105.8°—110°	41° —43.3°

The range of deviation from the normal within the limits of which life can be maintained for brief periods is comprised between 92° F. and 110° F. A temperature of 95° F. on the one hand or of 106° F. on the other, already indicates great danger, especially if it be prolonged, and beyond these limits in both directions the danger to life speedily becomes extreme.

(a) **Temperature of Collapse or Shock.** — A considerable and rapid fall of temperature attends the collapse which sometimes occurs during or towards the close of some of the essential fevers. In enteric fever this condition may be produced by hemorrhage, or by sudden peritonitis due to perforation, or in consequence of sudden failure of the heart. The last

of these accidents is liable to occur in any very grave case of fever, and occasionally follows the critical fall of temperature which occurs in pneumonia, relapsing fever, and more rarely in other febrile diseases.

Very low axillary temperatures are met with in the stage of collapse in the algid or cold stage of cholera, the internal temperature as indicated by the vagina or rectum remaining high. Great depression of the general temperature occurs in the collapse produced by various poisons, and especially by large quantities of alcohol. The temperature is apt to fall considerably below the normal in ordinary deep alcoholic intoxication, especially if the patients have been exposed to cold and wet.

(b) **Subnormal Temperature.** — This condition attends considerable losses of blood; starvation from any cause; the wasting of certain of the chronic diseases, such as cancer of various organs; some diseases of the brain and spinal cord and the later stages of chronic diseases of the lungs and heart, especially when accompanied by dropsy.

The temperature is very apt to reach subnormal ranges in the morning for a few days at the termination of febrile disorders.

(c) **Normal Temperature.**—If in the course of a continued fever, as enteric, the temperature, which has been elevated two or three degrees or more, *suddenly* falls to normal or near it, though not below, this in itself is significant of something wrong, and may even acquire the importance of the "temperature of collapse," as indicating internal hemorrhage, perforation, or failure of the heart.

(d) **Subfebrile Temperature.**—Slight elevations of temperature often accompany trifling and transient disturbances of the general health, especially in children. They are also observed at the beginning of gradually developing fevers, as enteric, and at the close of slowly subsiding febrile conditions. In obscure chronic cases they are of importance as indicating the existence of actual disease which may not manifest its ordinary symptoms.

(e) **Moderate Febrile Temperature.**—When the morning temperature reaches 101°–102° F. and the evening shows a further increase of one or two degrees, we have to do with actual fever. So long, however, as the temperature does not exceed these limits, there is no serious danger from the fever process itself.

(f) **High Febrile Temperature.**—When the temperature in the morning is above 102°–104° F. and in the evening reaches or ranges higher than 104.5°, the case becomes serious from the intensity of the fever alone, and active treatment becomes imperative. High fever is unattended by immediate danger to life if it be transient, but when prolonged it is ominous. A temperature of 105° or even 107° in the hot stage of an ague, when the whole attack lasts but a few hours, is much less dangerous than the same temperature occurring, even for a short time, in the course of one of the continued fevers, when the patient's powers of resistance are called upon to withstand some degree of fever for several days or weeks.

(g) **Hyperpyrexia, or Intense Febrile Temperature.**—The temperature reaches 105.8° and continues to rise, or at all events does not fall. The condition is one of extreme and imminent danger to life. The resources of the art of medicine are put to their severest test. Hyperpyrexia often

supervenes with great suddenness. Not a moment is to be lost. The most prompt and radical measures to reduce the temperature of the body too often fail to avert the fatal result. This condition has been encountered after injuries to the brain and to the upper part of the spinal cord; in lock-jaw; in sunstroke, and very often in the infectious diseases, especially scarlet fever and pneumonia. It sometimes occurs in rheumatic fever, especially after the intensity of the symptoms has begun to subside, or even when the patient is apparently almost well. Hyperpyrexia is often one of the indications of approaching death. Hence, in certain cases the futility of treatment. In such cases a temperature of 110° to 112° is sometimes seen. The temperature sometimes continues to rise slowly for an hour or two after death.

The thermometer may be made to indicate a temperature much higher than that of the patient's body, by friction, or by being slipped against a poultice or hot-water bag, or into a cup of tea, when the attention of the nurse is given to other duties. These tricks are sometimes played by hysterical girls. They are readily detected by repeated observations under the eye of the attendant. A number of cases have been recorded in the medical journals in which excessively high temperatures—120°, 150°, even 170° F.—have been noted and apparently verified by repeated and most careful observations. Many of the patients have subsequently been found to be very clever pretenders and tricksters, but the method by which the high temperatures have been recorded has not been explained. In such cases the temperature should be taken in several different regions, axilla, mouth, rectum, etc., at the same time, and the temperature of the urine when voided.

Transitory Variations.—The temperature of a fever patient may be somewhat affected by excitement, fatigue, or exposure. Hence hospital patients often show for a few hours after admission a temperature higher than subsequently, or, if they have been exposed to cold, lower than really corresponds to their condition.

It is a peculiarity of the state of convalescence from the acute fevers that the temperature, though normal, is disturbed by trifling causes, and may be made to rise two or three degrees by the first visit of a friend, the first solid food, or even by sitting up. Such rises are usually very brief, the temperature quickly falling again to normal. They occasion uneasiness lest they be the beginning of a relapse. On the other hand it occasionally happens that, though all the other symptoms have disappeared and the patient is almost well, the temperature remains subfebrile, and the patient is for that reason alone kept in bed. In such cases all traces of fever vanish upon cautiously allowing the patient to sit up an hour or so each day.

Surface Thermometry.

This method is of inferior value for diagnostic purposes. The bulb consists of a fine coil at right angles to the tube and forming an expanded base for it. Observations may be taken at the same time in corresponding positions on both sides of the body. The general temperature must be noted.

Normal surface temperature (Kunkel). Temperature of room 68° F.—20° C.

	F.	C.
Forehead	93.38–93.92°	34.1–34.4°
Cheek under the zygoma	93.92°	34.4°
Tip of ear	83.84°	28.8°
Back of hand	90.5 –91.76°	32.5–33.2°
Hollow of the hand (closed)	94.64–95.18°	34.8–35.1°
Hollow of the hand (open)	93.92–94.64°	34.4–34.8°
Forearm	92.66°	33.7°
Forearm (higher)	93.74°	34.3°
Sternum	93.92°	34.4°
Pectorales	94.46°	34.7°
Right iliac fossa	93.92°	34.4°
Left iliac fossa	94.28°	34.6°
Os sacrum	93.56°	34.2°
Eleventh rib (back)	94.1°	34.5°
Tuberosity of ischium	89.6°	32.0°
Upper part of thigh	93.56°	34.2°
Calf	92.48°	33.6°

The temperature of the skin is slightly higher over an artery than at some distance from it, over muscle than over sinew, over an organ in activity than when at rest, in the frontal than in the parietal region of the head, and on the left side of the head than on the right.

Local elevation above the general temperature has been noted on the surface of the head in cases of mania and meningitis. Local elevation of the temperature has also been observed in cerebral tumor and abscess. A local rise of temperature also occurs over the painful points in some cases of neuralgia and in areas of superficial inflammation. The surface temperature is increased in the region corresponding to the exudate in croupous pneumonia. Irregularly distributed areas of elevated surface temperature sometimes occur in hysterical persons.

Subnormal temperature may be observed in a limb from which the blood supply is cut off by the tourniquet or obstruction of the main artery, in an œdematous or cyanosed part, and in gangrenous areas. Weir Mitchell called attention to the effect of posture upon local temperature. He found the surface of the dorsum and sole of the foot 0.4° C. to 1° C. cooler in the erect than in the recumbent posture.

Charts.—The temperature must be recorded at once. At the same time a record of the pulse-beats and movements of respiration per minute is to be made. They are to be carefully counted while the thermometer is in position.

Ruled sheets, called "temperature charts," or "clinical charts," are sold in the shops for this purpose. The form here shown will be found very convenient. It may be so kept with little trouble as to preserve in a compact form all the important facts of an acute case, and is equally useful in hospital and in private practice. The ruled space is arranged for twenty-one days by vertical lines, the weeks being divided by heavy lines. The space for each day is again subdivided for the morning and evening record, as indicated by the M and E. At the left margin the purposes of the spaces formed by the transverse rulings are indicated. At the top the number of movements of the bowels; immediately below the quantity of urine passed, which may be recorded in fluidounces or cubic centimetres; then the scale of *Fahrenheit*, with the equivalent *Centigrade* opposite on the right

margin. The coarse horizontal line at 98.4° F. indicates approximately the normal. At the bottom are, first, spaces for each day of the disease, then similar spaces divided by a diagonal line for morning (upper, left triangle) and for the evening (lower, right) pulse-rate; below these again corresponding spaces for the respiration-rate, and at the bottom of the chart spaces for the date or day of the month.

Important clinical facts, as "hemorrhage," "convulsions," "suppression of urine," etc., may be noted at the time of their occurrence between the vertical lines on the right or upper side of the chart in the position indicated by the arrows, under the words "clinical memoranda."

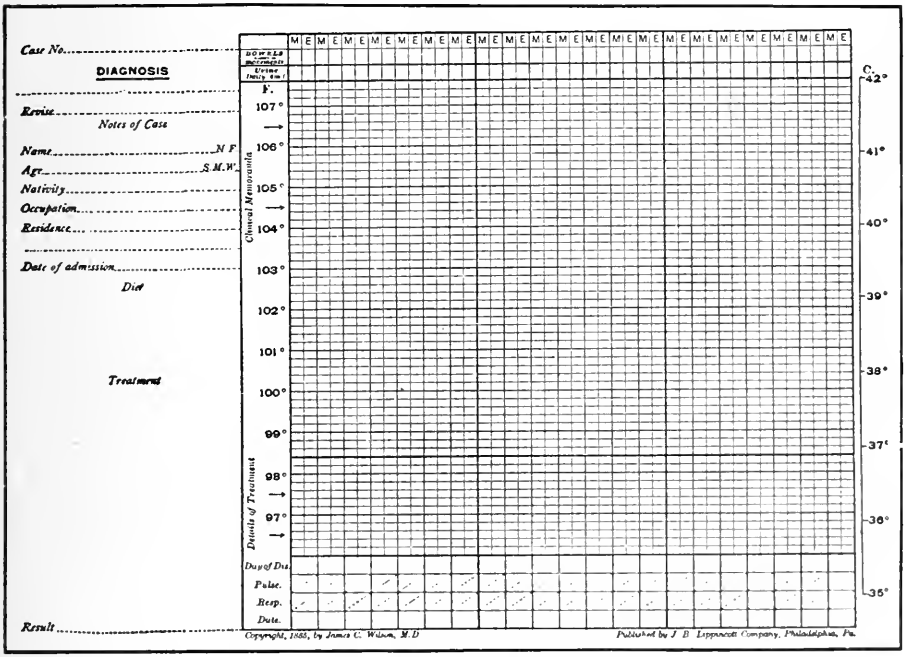


Fig. 29.—Clinical chart. Actual size 28 x 21 cm.

While changes in treatment, and in particular such temporary changes as are made necessary by accidents, like hemorrhage, convulsions, or suppression of urine, may be noted at the left or lower side under the words "details of treatment," as shown by the arrows.

The previous history and the condition of the patient when first seen may be written on the back of the chart.

The spaces corresponding to a degree of the *Fahrenheit* scale are divided into fifths. The temperature, as observed, is designated by a dot in the appropriate position. These dots joined by ruled lines form a zigzag line, called the temperature curve. It is usual to form the general curve of the case by means of the regular morning and evening temperatures, and to indicate the result of observations made at other hours by dots in the appropriate positions, with figures and letters showing the hour at which they were made; thus, 12 noon, 3 P.M., or 6 A.M.

It is customary to join the general curve or range by lines drawn with black ink; the hourly or three-hour observations by lines drawn with red ink. If the fever be prolonged beyond three weeks two or more charts may be pasted together. These charts thus kept are not only of value for preservation: they are also of immediate use as showing at a glance and with precision the facts of the case at every period from its coming under observation, the course it is running by a comparison of the symptoms day by day, and in a general way the effects of treatment, the changes of which are fully presented. Especially are they valuable in fevers in enabling us to watch the course of the temperature, which is a conspicuous part of the natural history of the disease and conforms in most of the acute infections to a type not only in its daily fluctuations but also in its duration.

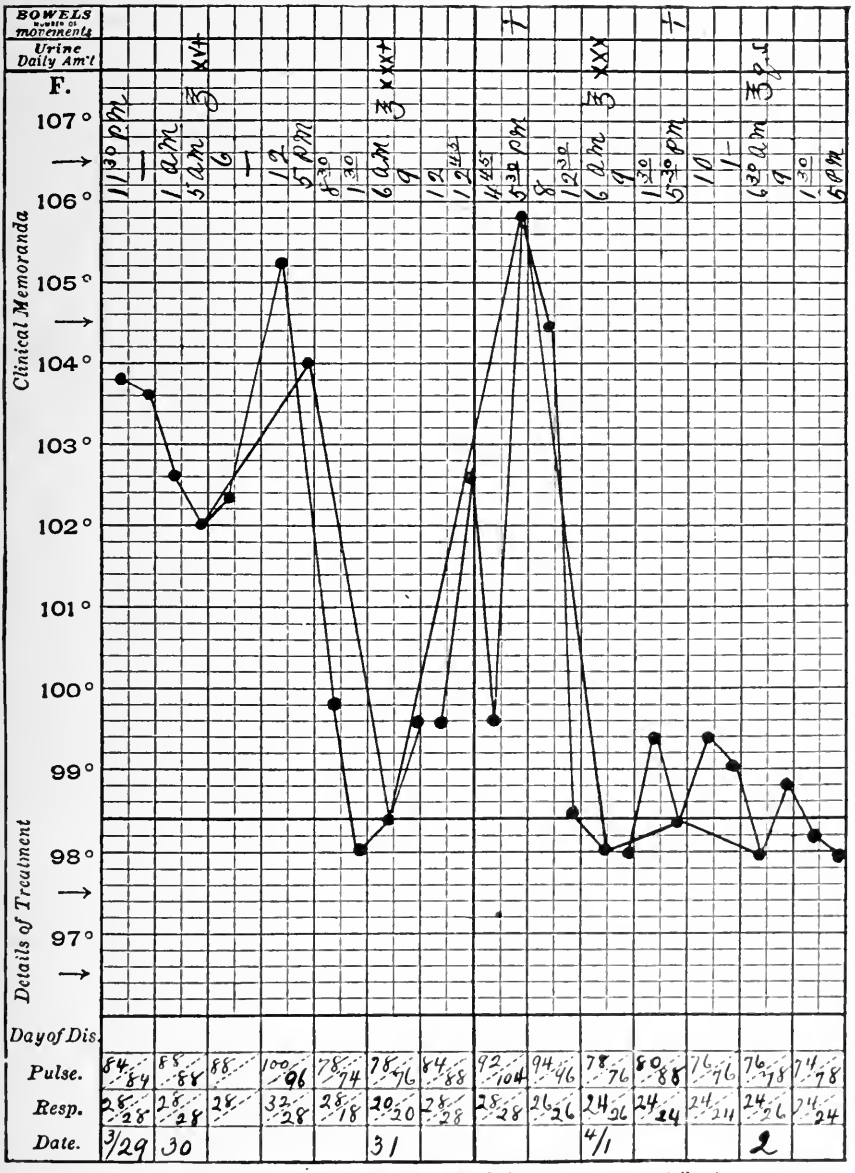


FIG. 29a.—Abscess of the Prostate—operation refused. Fall of temperature following spontaneous rupture.—Pennsylvania Hospital.

II.

PHYSICAL DIAGNOSIS.

General Considerations.—Physical diagnosis is the method of discriminating diseases by the direct aid of the special senses, namely the eye, the ear, the touch. The diagnostic criteria thus obtained are known as physical signs. They depend upon the physical nature and structure of the organs or parts examined and vary with the changes caused by disease. Hence they are divided into two groups—normal or healthy, and abnormal or morbid physical signs. As they bear a direct relation to the anatomical condition of structures, their form, contour, density, elasticity, and so forth, and similar physical conditions may be present in different diseases, and as morbid processes may arise in the absence of perceptible alterations in parts, it is evident that physical signs taken singly are not diagnostic of particular diseases. They reveal the anatomical condition but not the morbid process causing that condition, and attain their full value in diagnosis only when considered in relation to other signs and symptoms and the clinical history of the case.

Pathognomonic signs are those supposed to be diagnostic of particular diseases. In view of the facts just mentioned, the use of the term pathognomonic in this sense is erroneous in theory and misleading in practice. A physical sign is the manifestation of a normal or morbid physical condition, not of health or disease. It is most important for the student to bear this fact clearly in mind. Signs at one time regarded as pathognomonic, as for example the crepitant râle in pneumonia, are now known to occur in other conditions, as œdema of the lungs and partial atelectasis.

Physical diagnosis is constantly employed in the study of general maladies and in local diseases of all parts of the body, but it is of special service in the investigation of diseases of the respiratory and circulatory organs.

Methods.—The methods of physical diagnosis are INSPECTION, PALPATION, MENSURATION, PERCUSSION,—including RESPIRATORY PERCUSSION, PALPATORY PERCUSSION, and AUSCULTATORY PERCUSSION.—and AUSCULTATION. In the examination of patients these methods are used systematically and in succession, the signs elicited by one serving to confirm, extend, or control the knowledge obtained by the others.

Technic.—The physical examination must under all circumstances be conducted in a routine manner. More errors in physical diagnosis arise from want of system than from want of knowledge. When the patient is in bed, the bared chest, abdomen, and back must be in turn examined by the several methods. Under some circumstances a towel or a single thickness of raiment may be used as a covering, especially in percussion and immediate auscultation. For inspection and palpation the surface should be bared.

The same rules apply to the examination of ambulatory patients. In all cases the outer clothing should be removed. Physical signs cannot be elicited through heavy clothing, starched linen, or the corset; while

silk, stiff shirt fronts and the braces cause upon deep respiratory movements crackling and friction sounds that have nothing to do with the organs within the chest. In all cases the examination must be conducted with tact, judgment, and due regard for the sensibilities of the patient.

The recognition of abnormal physical signs involves a familiar knowledge of those which are normal and their variations within the bounds of health and of the anatomy and physiology of the organs or parts examined. Equally necessary is a ready knowledge of the pathological changes upon which abnormal signs depend. The skilled diagnostician cultivates the habit of seeing with his mind's eye the changes in structure caused by disease. A long apprenticeship in the post-mortem room is an essential preparatory course for good work at the side of the bed.

INSPECTION.

This method of physical diagnosis is of the widest application in the study of disease. In many cases a provisional, in some a positive diagnosis may be made upon a careful study of the external clinical phenomena by inspection alone. The facies hepatica, emaciated neck and limbs, and enormously distended abdomen in cirrhosis of the liver, the enlarged parotids, disfigured countenance, and projecting lobule of the ear in mumps, the unilateral flushing and jerky dyspnœa in croupous pneumonia, and the rash in the eruptive diseases, tell their own tale. In a narrower and more technical sense inspection is especially of value in the diagnosis of diseases of the thoracic and abdominal organs.

The clothing must be removed. The light must be good. The skilled diagnostician makes use now of direct light, by which extensive surfaces are fully illumined; now of oblique light, by which local elevations and depressions are accentuated and pulsations are marked by moving shadows.

By inspection we obtain information in regard to the size, form, or contour, the appearance of the surface, and the movements of the thorax and abdomen.

Inspection of the Thorax.

The Size.—The size of the chest is determined by the volume of its contents. Within the limits of health there are wide variations. A sedentary life tends to shallow breathing and small lungs. The chest then conforms to the inspiratory type. The anteroposterior diameter is short, the upper intercostal spaces wide, the lower narrow, the costal angle acute. We speak of such a chest as shallow. Active, out-door occupations favor habitual deep breathing and increase in the size of the lungs. The chest now conforms to the expiratory type. The anteroposterior diameter is relatively long, the upper intercostal spaces narrow, the lower wide; the costal angle is obtuse. Such persons are deep chested. Diseases which diminish the size of the lungs, as chronic tuberculosis and fibroid phthisis, correspondingly reduce the size of the thorax, while so-called pseudo-hypertrophic emphysema greatly increases its size. But these changes are accompanied by definite changes in form. Excessive subcutaneous fat sometimes gives rise to an apparent increase in the size of the chest.

The Form.—The form of the chest varies with its size. In infancy and early childhood it is somewhat cylindrical,—that is, its anteroposterior diameter and its transverse diameter are nearly the same,—and the respiration is chiefly diaphragmatic. In adults the cross section of the trunk is oval and symmetrical. Upon deep inspiration the anteroposterior diameter of the chest is increased; on forced expiration it is diminished.

Deformities.—The general deformities in childhood are commonly due to respiratory obstruction in the upper air-passages, as from adenoid growths in the nasopharynx, enlargement of the tonsils; or in the lungs, as in bronchopneumonia or phthisis. Rickets plays an important part. In adult life they are commonly caused by fibroid changes in the lungs, pulmonary tuberculosis and emphysema. Unilateral and local deformities are caused by pleural effusions, the retraction which follows the resorption or removal of such effusions, hypertrophy of the heart, and aneurismal or other intrathoracic tumors. These abnormal modifications in form are more marked when they occur early in life. The following deviations in form are to be considered:

Fig. 30.—Alar deformity of chest.—German Hospital.

(a) **The Alar or Pterygoid Chest.**—The chest is unnaturally small and narrow. The mesial borders of the scapulæ project like budding wings, the ribs are extremely oblique, the shoulders droop, the neck and chest appear preternaturally elongated, the head is carried unduly forward, and the costal angle is acute. This form of chest is sometimes described as the "paralytic chest." Persons suffering from pulmonary tuberculosis frequently present this form of chest, but it may also occur in poorly nourished individuals who are not phthisical.

(b) **The Rhachitic Chest.**—The sternum may project, giving rise to the deformity known as *pigeon breast*. The sides of the chest are flattened and curve forward to the prominent sternum, as the sides of a boat to the keel—*pectus carinatum*. From the base of the ensiform cartilage a broad shallow depression or groove passes downward and outward to the infra-axillary region—Harrison's furrow. In some instances the cartilages of the ribs lose their curve and become straight, causing the chest to be quite flat in front instead of being rounded. In others there is a shallow

longitudinal groove on each side of the front of the chest, a little external to the sternum and nearly parallel to it. The remarkable deformity known as FUNNEL BREAST sometimes but by no means always is due to rickets. It consists in a deep and rather abrupt crater-like depression in the region of the base of the ensiform cartilage. Not rarely there may be felt and sometimes seen a line of nodular thickenings along the chondrocostal articulations on each side, known by the fanciful name of *the rhachitic rosary.*

(c) **The Barrel Chest.**—The deformity characteristic of emphysema is very striking. The antero-posterior diameter is greatly increased. The thorax is in a state

FIG. 31.—Paralytic chest.

FIG. 32.—Funnel-shaped deformity of chest. —Jefferson Hospital.

of distention greater than that produced in health by the deepest inspiration. It is arched before and behind. The manubrium and body of the sternum are sometimes bent at an angle—ANGULUS LUDOVICI. The shoulders are high, the neck short, and the costal angle very obtuse. Dorsal kyphosis due to the carrying of burdens upon the shoulders, to advancing years, or to vertebral caries may simulate the barrel-shaped chest of emphysema.

(d) **Deformities of the Spine.**—Curvatures and twisting are very common. The slighter forms are often overlooked. They may be recognized upon careful inspection of the bare back, the spinous processes being marked by a dermatographic pencil. Marked curvatures in which rotary displacements are prominent derange the relations of the thoracic viscera to the bony landmarks and render the physical examination of the chest

difficult and the signs uncertain. The cardiac impulse may be displaced upward or to the left; abnormal bulging may simulate aneurismal or other intrathoracic tumor and areas of atelectasis with compensatory emphysema occur. Abnormal rigidity of the spine may be due to spastic contraction of the muscles in Pott's disease or to spondylitis deformans. When ankylosis has developed the spine is persistently rigid. These signs may be recognized upon attempts to bend forward or backward or to rotate the shoulders while the pelvis is held fixed. An examination of the vertebral column forms part of every routine examination of the chest.

FIG. 33.—Emphysematous type of chest.—German Hospital.

Unilateral changes in the shape of the chest consist in diminution and enlargement.

Unilateral Diminution.—Flattening of one side of the chest is a sign of chronic pulmonary tuberculosis of the corresponding lung, fibrosis of one lung, or a pleural effusion which has undergone resorption or been cured by operation. The circumference and anteroposterior diameter are diminished; the bilateral diameter is increased; the side is angular and flattened before and behind; the upper intercostal spaces are widened, the lower narrowed; the shoulder is lowered and there is lateral curvature of the spine, the convexity being towards the opposite side. The vicarious enlargement of the sound lung gives rise to marked differences in the circumference of the two sides. When the deformity is due to tuberculous disease of the upper lobe, the flattening is more marked in the upper region of the chest; when to old pleurisy it is more marked at the base. Unilateral flattening of the chest is attended by pleural adhesions. If obstruction of the main bronchus occurs in childhood, the resulting collapse of the lung may cause an acute unilateral flattening of the chest. Lateral spinal curvature may simulate diminution of the chest from pulmonary disease.

Unilateral Enlargement.—This deformity of the chest is a sign of vicarious enlargement of one lung as a result of chronic disease of its fellow, pleural effusion, large hæmothorax, pneumothorax, and rarely of rapidly growing malignant disease. Pseudohypertrophic emphysema may in rare instances involve one lung when the other has undergone fibroid changes in consequence of previous disease. The enlarged side is rounder than the other; its anteroposterior diameter longer; the intercostal spaces wide;

the shoulder raised and the spine curved laterally, the dorsal convexity being towards the enlarged side.

The foregoing alterations in the form of the chest are very obvious when the physician stands behind the seated patient and looks obliquely over his shoulders and the front of his chest.

Intercostal Spaces.—In large pleural effusions and in pneumothorax the normal depression of the intercostal spaces is obliterated and the surface smooth as contrasted with the opposite side. Bulging of the intercostal spaces is rare. It may be seen at the base of the chest in large empyema of long standing.

Fig. 34.—Deformity following the resorption of a pleural effusion.—German Hospital.

Local Changes.—Local changes in shape consist in (a) circumscribed retraction or (b) prominence.

Local retraction is a sign of the following conditions:

Tuberculous Consolidation of a Portion of the Lung.—This is usual at the apex and most obvious in the supra- and infraclavicular regions. It is attended by pleural adhesions.

A Superficial Cavity.—Circumscribed depressions due to this cause are often seen on the anterior surface of the chest near the sternal border and extending over one or two intercostal spaces. Flattening in the posterolateral aspect of the chest opposite the spine of the scapula and below its level is sometimes seen in pulmonary abscess.

Old Pleurisy.—A broad, shallow depression in the anterolateral region at the base of the chest is common after pleural effusions. The funnel breast sometimes follows unilateral pleurisy. This deformity in shoemakers has been attributed to the pressure of the last against the breastbone.

Local retraction in children may follow croup, bronchopneumonia, and rickets. The deformities caused by these agencies are symmetrical and have already been considered.

Local prominence is a sign of circumscribed pleural effusion, large vomicæ when distended with fluid, diaphragmatic hernia when congenital, tumor of the lung or of the chest wall, mediastinal tumor, abscess of the chest wall, and empyema necessitatis. In a considerable proportion of healthy persons that region of the chest wall which overlies the heart—*the precordial space*—is slightly prominent. In children and occasionally in adults prominence of the precordial space results from cardiac hypertrophy or pericardial effusion. The bulging occupies the space between the third and seventh costal cartilages on the left side and the left midclavicular line and the sternum. It may extend to the right nipple. Aneurism of the arch of the aorta causes local bulging of the chest wall and in rare instances aneurism of the descending aorta may erode the ribs and give rise to a circumscribed tumor of the dorsal region to the left of the spine. Inspection of the back frequently reveals local prominences of importance in diagnosis. A sharp projection of the spinal processes occurs in vertebral caries. The mesial borders of the scapulæ stand out prominently in the pterygoid chest. A congenital rounded tumor in the middle line, translucent and partly reducible, is the sign of spina bifida. This defect of development is frequently associated with other deformities, as hydrocephalus or club-foot. A dusky-red, brawny swelling, commonly in the cervical region, discharging pus from several sinuses, is a carbuncle. It occurs frequently in diabetes mellitus, and in all cases the urine should be examined for sugar. Rounded or lobulated elastic tumors, painless and usually movable, are fatty. They sometimes so closely resemble abscesses as to require aspiration for the differential diagnosis. Abscesses appear as fluctuating swellings as the result of caries of the vertebræ, usually tuberculous, and may burrow in various directions. I have seen a large, oblong tumor to the left of the dorsal spine formed by an aneurism of the descending aorta, and a similar tumor in the left lumbar region which pulsated and was connected with a left-sided empyema. In disseminated sarcoma of the skin the lesions are common on the back, appearing as circumscribed nodular masses varying in size from a small shot to a walnut, dark in color and mostly movable.

Fig. 35.—Aneurism of the descending thoracic aorta; perforation of chest wall.—Penna. Hospital.

Surface.—The appearance of the surface of the chest only exceptionally yields, upon inspection, physical signs of importance. We note emaciation or an abundant panniculus adiposus, jaundice, cyanosis and pigmentation, the eruptions and scars of the exanthemata or of syphilis or other chronic disease, enlargement of the superficial lymph-nodes at

the root of the neck and in the armpits, patches and lines of dilated venules and dilated and tortuous venous trunks. The appearance of linear patches of herpes in shingles—zona, herpes zoster—in the course of the intercostal and lumbar nerves, often clears up the diagnosis where there has been severe burning pain upon one side of the chest or abdomen.

The Movements of the Chest. — Normal and abnormal types of respiration will be considered in a subsequent section. Anomalous movements that affect both sides of the chest occur in dyspnœa, inspiratory dyspnœa, expiratory dyspnœa, Cheyne-Stokes respiration, exaggerated thoracic, exaggerated abdominal breathing, and so on.

Abnormally deep respiration in the absence of any apparent difficulty either in inspiration or expiration is seen in diabetic coma.

In the emphysema of the aged and in earlier life in some cases of hereditary syphilis and pulmonary tuberculosis, calcification of the costal cartilages and associated changes in the ribs cause the walls of the chest to move through a limited space as a whole—*en cuirasse*.

Unilateral modifications of the respiratory movements may consist of (a) diminished expansion of one side or (b) increased expansion of one side.

(a) Diminished expansion of one side may involve the entire side, as in large pleural effusion, pneumothorax, pneumonia involving the whole of one lung, tuberculous consolidation of a lung, or tumor of the lung or pleura. The affected side is not only immobile but it is also distended and altered in contour. In tuberculosis it is, however, usually contracted in consequence of pleural adhesions and sclerotic changes in the lung. In massive pneumonia it is almost immobile but not enlarged. Contraction also occurs in the occlusion of a large bronchus from the presence of an aneurism or other tumor.

In tuberculosis confined to the apex of one or both lungs there is failure of expansion in the corresponding region of the chest.

Diminished unilateral expansion may be a sign of infradiaphragmatic disease—on the right side, of an enlarged liver or hepatic tumor; on the left, of an enlarged spleen or tumor in the splenic region.

In rare instances a hemiplegia or paralysis of one side of the diaphragm or a diaphragmatic hernia may be the cause of diminished expansion of one side of the chest.

Non-expansive inspiration is attended with retraction of the interspaces. This sign is especially noticeable in the inframammary, the infraaxillary, and the infraclavicular regions in partial atelectasis or collapse of the lungs, in obstruction of the glottis as in pseudomembranous laryngitis, œdema of the glottis, or pseudomembranous bronchitis such as occurs in infralaryngeal diphtheria or in the diffuse atelectasis of bronchopneumonia. Under those conditions both sides are involved. When a main bronchus is occluded the sucking in of the intercostal spaces upon inspiration is limited to the affected side. This phenomenon is caused by intrathoracic negative pressure during inspiration, in consequence of which the soft parts of the thoracic wall yield to the external pressure of the atmosphere.

(b) Increased expansion of one side of the chest is usually compensatory. It occurs when the respiratory movement of the opposite side

is interfered with by pathological conditions of the lung, as tuberculosis, pneumonia, fibrosis and atelectasis from other causes, or by pleural effusion, pneumothorax, or tumor, and thus becomes a sign of those conditions.

The Diaphragm Phenomenon — Litten's Sign. — The diaphragm approaches the wall of the thorax in expiration and comes into contact with it at the end of the act. It is separated or *peeled off* from it in inspiration. These movements are rendered visible by the procedure suggested by Litten in 1892. The patient is placed upon his back with his chest bared and his feet toward a window. Cross lights are excluded. If the examination is made at night, a strong light held at the foot of the bed serves the purpose. The observer stands at a little distance and views the surface of the lower part of the chest obliquely. Upon deep inspiration a short, narrow, horizontal shadow is seen to move from the sixth intercostal space downward over two or more interspaces upon both sides. During expiration this shadow moves up again to the line from which it started but is less distinct. It may in some cases be seen in the epigastrium. This phenomenon is practically present in all healthy persons, the only exceptions being due to abnormal thickness of the chest walls and inability on the part of the patient to make full, deep respiratory movements. It is best observed in young, lean, muscular persons. The extent of the movement of the shadow in normal chests is about two and a half inches; upon forced breathing slightly more than this.

The descending shadow is due to the undulation of the chest wall caused by the separation of the diaphragm from its contact with the lower part of the thorax and the descent of the border of the lung into the wedge-shaped space between them during inspiration, and the reverse shadow by the retraction of the lung and the coming together of the diaphragm and chest wall during expiration.

The shadow is absent upon the affected side in pneumonia of the lower lobe, pleural effusion, extensive pleural adhesions, intrathoracic tumors, and marked emphysema. In these conditions the diaphragm does not approach and recede from the chest wall and the undulations which cause the shadow do not occur. The extent of the movement is lessened in conditions of debility, slight emphysema, and upon the affected side in phthisis. In the latter condition there are probably two factors in restricting the movement, diminished pulmonary expansion and limited pleural adhesions.

Litten's sign is present in hepatic and splenic enlargements and in subphrenic abscess and may be of service in the differential diagnosis between those conditions and pleural effusion. In very large ascites it may be absent.

The Movements of the Heart. — Inspection yields important physical signs in regard to the heart and great vessels in health and disease. These signs relate to (a) the cardiac impulse; (b) other movements of the surface having the cardiac rhythm: (1) pulsations at the root of the neck, (2) aneurism, (3) tumors in contact with large arterial trunks, (4) pulsating empyema.

(a) **The Cardiac Impulse.** — With the systole of the heart there is seen in most normal chests an outward movement or pulsation in a limited

area in the fifth left intercostal space just beyond the parasternal line—the visible impulse or so-called apex-beat of the heart. In infants and young children, owing to the proportionately greater size of the liver, the impulse is often visible as high as the fourth interspace, while in aged persons it may normally be as low as the sixth interspace. It is occasionally absent in healthy persons, especially those having deep chests and capacious lungs. It invariably takes place at the time of the contraction of the ventricles. The most important factor in the production of the impulse is the change in the direction of the long axis of the ventricles against the resistance of the chest wall. It is a mistake to speak of it as a "blow" or "impact" against the wall of the chest, since that part of the heart which causes it, namely, the apex of the right ventricle, is already in contact with the wall in diastole and simply becomes more tense and prominent during systole. Around the point where the soft parts are protruded by the impulse they are very slightly retracted at the time of its occurrence—the "*negative impulse.*" This is due to the lessening size of the contracting ventricles, which, being air-tight within the cavity of the chest, must be followed down under the pressure of the atmosphere by the elastic and yielding lungs and the somewhat yielding intercostal tissues. A clear conception of this fact renders intelligible the systolic recession of the chest wall occasionally seen in emaciated persons in the third, fourth, or even the fifth intercostal space, close to the left border of the sternum.

Since the normal impulse is caused by the apex of the right ventricle and not by that of the left, which extends further downward and is separated from the wall of the chest by a tongue-like projection of the lower lobe of the left lung, the apex of the right ventricle is sometimes spoken of as the "clinical apex" and that of the left ventricle as the "anatomical apex" of the heart.

The normal impulse is usually limited in extent, often not exceeding an inch square. Its position varies somewhat with the posture. When the patient lies upon the left side, it may shift an inch or more towards the axillary line, and a similar displacement to the right, but less in extent, takes place when he lies upon the right side. The impulse is less marked and less extensive in the recumbent than in the erect posture. These changes in the position of the heart are caused by corresponding alterations in the position of the apex under the influence of gravity. The position of the impulse is little influenced by quiet breathing, but as the diaphragm sinks and the lower ribs are elevated in inspiration a change in the relation of the apex-beat to the chest wall, in some instances amounting to an interspace, may be observed upon forced breathing.

The impulse becomes forcible and extended when the normal heart is acting rapidly and with force under physical or mental stress and in thin, nervous persons, and it is often extended in young children even at rest.

The character of the impulse and its extent are best studied by palpation, but inspection alone enables us in many cases to determine that the impulse is extended, heaving, tapping, or undulatory.

Displacements of the Impulse of the Heart.—*Displacements due to Changes in the Heart Itself.*—The impulse is displaced downward and toward the left in hypertrophy and dilatation of the heart, and the combi-

nation of these conditions is the most common cause. Enlargement of the left ventricle tends to displace the visible impulse downward, enlargement of the right ventricle tends to displace the impulse to the left, and both of these conditions tend to increase its extent.

Pressure Displacements are next in Order of Frequency.—The heart is dislocated upward in pressure from below the diaphragm, as in excessive tympany, ascites, massive tumors, large cysts, and pregnancy. In any of these conditions the impulse may be seen in the fourth interspace and to the left of the midclavicular line. The heart in pleural effusion, pneumothorax, or rapidly growing malignant tumors of the pleura, is displaced towards the opposite side. When these conditions are left-sided the impulse may disappear behind the sternum or become visible at its right border, or in extreme cases in the right nipple line. In large right-sided effusions, on the contrary, the impulse may be displaced as far left as the line of the anterior axillary fold. Cysts and abscess in the right lobe of the liver may displace the heart somewhat to the left and shift the impulse to a corresponding extent.

The heart may be displaced downward by an aneurism of the arch of the aorta or a mediastinal tumor. Under those circumstances the impulse is not only lower than normal but it is also somewhat further to the left.

Traction displacements of the heart occur in pulmonary cirrhosis and long-standing disease of the pleura. The displacement is toward the affected side. Pleuropericardial adhesions and negative pressure constitute the mechanical factors by which this group of displacements is brought about. A cardiac impulse may be seen to the right of the sternum, or to the left of the left midclavicular line, or if there be great retraction of the upper lobe on either side there may be visible cardiac pulsation at the corresponding border of the manubrium. Spinal curvatures and rotations may produce such displacement of the heart as to cause a wholly abnormal position of the visible cardiac impulse or its absence altogether. Dextrocardia may be the cause of a right-sided impulse, an anomaly also present in complete transposition of the viscera.

Systolic Retraction. — I have already spoken of the negative impulse present under normal conditions in the immediate proximity of the apex-beat and the more extended systolic recession of the interspaces occasionally seen along the lower sternal border. These are distinctly accentuated when a hypertrophied and somewhat dilated heart is acting forcibly. As these forms of systolic recession are due to atmospheric pressure, they may be spoken of as *pulsion recessions* in contradistinction to those due to the drawing in of the surface in consequence of adhesions, which may be called *traction recessions*.

The latter are seen in adherent pericardium with chronic mediastinitis. The impulse is undulatory and in the region of the apex there is marked systolic retraction. Owing to the enlargement of the heart the precordial region is prominent and the chest asymmetrical. The impulse is greatly extended.

Broadbent's Sign.—When the heart is extensively adherent to the diaphragm, there occurs with each pulsation a systolic tug. This may be communicated through the diaphragm to the points of its insertion in the wall of the chest and well seen in the eighth and ninth intercostal spaces in

the parasternal line; but Broadbent has pointed out the fact that it is often also seen on the left side behind, between the eleventh and twelfth ribs. Careful inspection in this region will frequently reveal a systolic retraction of the chest wall, which becomes more evident upon deep inspiration.

Visible Pulsations of the Heart in Regions other than the Apex.—These are mostly due to retraction of the lungs. In debilitated and bed-ridden persons and especially in the graver forms of anæmia, the breathing is shallow and the lungs are not fully expanded. Their borders are therefore more or less withdrawn from the space which they normally occupy between the heart and the chest wall. The pulsations of the conus arteriosus and right ventricle thus frequently become visible in the second, third, and fourth left interspaces near the sternal border. In some instances these pulsations may also be observed to the right of the sternum. Such pulsations are also seen when the borders of the lungs are retracted as the result of fibroid phthisis.

(b) **Other Movements of the Surface of the Chest having the Cardiac Rhythm.**

1. Pulsations at the root of the neck will be described and their significance as physical signs pointed out in a subsequent section. They are venous and arterial.

Prominence of the veins of the neck is observed in emaciated and elderly persons otherwise in health. These veins are more or less distended upon expiration, particularly when cough occurs or dyspnœa is present. Transient engorgement results from efforts at lifting or from straining. Pathological conditions that give rise to engorgement of the jugulars are aneurism, mediastinal tumor, adhesive mediastinitis, and obstruction to the pulmonary circulation from any cause. Respiratory engorgement and collapse of the jugulars are especially marked in the dyspnœa of asthma and emphysema.

Collapse of the jugular upon one side, not disappearing when pressure is made upon it immediately above the clavicle, is a sign of thrombosis of the lateral sinus.

Pulsating Jugulars. — The pulsations are best studied on the right side of the neck and during quiet breathing. Pulsation communicated from the underlying carotid may be recognized by emptying the vein by stripping it upward gently with the finger-nail or the blunt edge of the tongue spatula. It does not refill from below.

The visible pulsations in the carotids often seen in thin, nervous persons without disease of the heart are without clinical importance. Violent throbbing of the carotids is common in aortic regurgitation and frequently occurs in simple hypertrophy of the heart without valvular lesions.

2. Aneurism.—Careful inspection of the anterior surface of the chest must be made in all cases of suspected aneurism. Direct and oblique illumination must be in turn employed, and the examination must be so conducted that profile views are made from above, the patient being in the sitting posture, and from the side, the patient being recumbent. In this way slight pulsations and pulsating prominences may be discovered. The pulsation of aortic aneurism is commonly present in the first and second

right interspaces near the sternal border and is sometimes accompanied by slight systolic elevation of the inner end of the clavicle. When the innominate is involved the pulsation may be seen at the root of the neck upon the right side or at the notch of the sternum. Aneurismal pulsations sometimes occur to the left of the manubrium sterni and elsewhere in the chest and are to be sought for in every doubtful case. Aneurisms that have perforated the chest wall appear as circumscribed globular or irregular pulsating tumors, the overlying skin being thinned and adherent and ultimately ulcerated, so that there is superficial clot formation and more or less continuous oozing of blood. The tumor may be soft and fluctuating; more commonly in consequence of the deposition of stratified fibrin layers within the sac it is dense and resistant. In the former case the pulsation is expansile; while in the latter case it is apt to be non-expansile, but forcible and heaving.

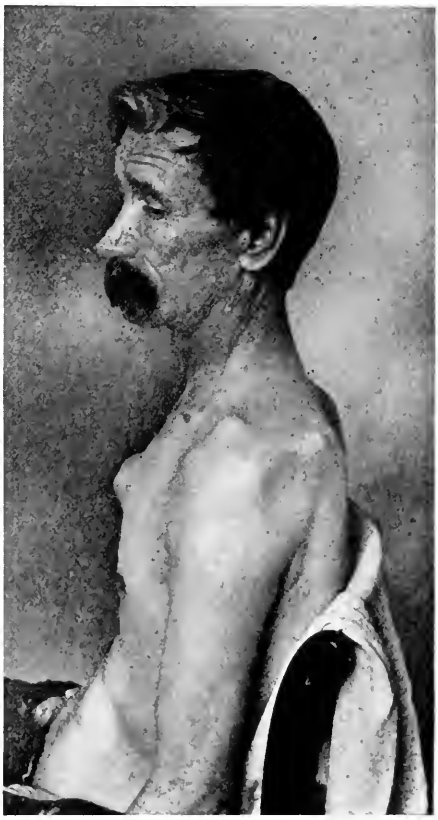


FIG. 36.—Aneurism of the thoracic aorta.—German Hospital.

3. TUMORS IN CONTACT WITH LARGE ARTERIAL TRUNKS.—Enlarged lymph-nodes, especially when single, and neoplasms in the neck overlying the carotid artery sometimes move synchronously with the pulsations of the vessel and present superficial resemblances to aneurisms. The tumor is dense, the pulsation not expansile, and other signs of aneurism are lacking.

4. PULSATING PLEURISY.—In neglected purulent effusions a pulsating movement synchronous with the cardiac rhythm is sometimes observed. The cases are not numerous. The phenomenon is almost always associated with left-sided effusions and occupies an extensive area of the lower anterolateral surface of the left chest. In cases where it is circumscribed and confined to the precordial region the differential diagnosis relates to aneurism and is attended with difficulty. A limited number of cases have been right-sided and in one or two of the reported instances the effusion has been serofibrinous. Pulsating empyemata may be intrapleural or the pulsations may occur in the extrapleural *empyema necessitatis*. None of the explanations of the mechanism by which the cardiac impulse in these cases is transmitted through the pus collection to the surface of the chest is satisfactory. An important factor is superficial ulceration of the costal pleura with loss of tone in the intercostal muscles.

Inspection of the Abdomen.

In the examination of this portion of the body the patient should be in the recumbent position and preferably in bed. The abdomen in exposed from the arch of the ribs to the suprapubic region. The patient should lie straight and flat. The head should be at first low and the lower limbs extended; later the head should be raised upon pillows and the thighs and knees strongly flexed, the heels being drawn up towards the buttocks, in order to relax the abdominal wall; finally it is often necessary to have the patient assume the standing posture, in which case the clothing or a sheet is supported about the hips by the patient or an assistant. The light must be good and the examination made from above, from the sides and obliquely. Physical signs of importance, such as asymmetry in contour or movement or slight local elevation or depression of the surface, may often be detected when otherwise not very obvious, if the observer stands at the patient's head and views the abdomen obliquely from above downwards. Combined inspection, palpation, and percussion are necessary. Auscultation is of inferior value in the examination of this region.

(a) **The Normal Abdomen.** — In infants and young children the abdomen is relatively larger as compared with the size of the chest than in adults. It is also more protuberant than in well-formed adults. It is larger in women than in men and is enlarged and protuberant in obese and elderly persons. In thin women who have borne many children it is relaxed, coarsely wrinkled, and pendulous. Tight corsets cause bulging of its lower segment. Transient prominence of the upper segment may sometimes be observed after a hearty meal.

The size of the abdomen in health varies greatly in different individuals according to the amount of subcutaneous and omental fat and the size of the intestines, which are apt to be distended in persons who habitually eat large quantities of coarse food. The physiological enlargement of the abdomen in pregnancy is frequently enormous.

The normal abdomen is symmetrical in contour, slightly arched from above downward and from side to side, the curves being more prominent, especially in the lower part, in the erect than in the recumbent posture. The navel is shallow and marked by irregularly spiral folds of skin in thin persons and deep and funnel-shaped in those who are fat.

The skin of the abdomen in healthy persons is opaque and the superficial veins are not conspicuous. In brunettes regularly distributed areas of increased normal pigmentation are present in the median line and above the flexures of the thighs. This coloration is deepened and conspicuous in pregnancy—*chloasma uterinum*. The respiratory movements of the diaphragm are communicated to the upper portion of the abdomen, the ensiform cartilage and the arch of the ribs being elevated and becoming more prominent with inspiration. In persons with very thin and relaxed abdominal walls the peristaltic movements of the stomach and intestines may be occasionally seen.

(b) **Inspection of the Abdomen in Disease.** — We study the size of the belly as manifest in general or local retraction or distention, alteration in form and contour, the appearance of the surface and abnormal move-

ments. In this connection the general rule that the size of a hollow anatomical structure or viscus varies with the contents must be borne in mind.

General Retraction of the Abdomen. — When the longitudinal and transverse curves of the surface are reversed and become concave instead of convex, the abdomen is described as scaphoid or boat-shaped. Two factors may cause this condition and they are frequently combined, namely, extreme wasting and irritative tonic spasm of the abdominal walls. The former occurs in actual starvation; inanition from any cause, especially malignant disease of the larynx or œsophagus, stricture of the latter from other causes, stricture of the pylorus without marked gastric dilatation, diabetes, phthisis, cerebrospinal fever, cholera, chronic diarrhœa, anorexia nervosa, and the pernicious vomiting of pregnancy; the latter, in meningitis, cerebral tumor, and lead colic, and especially when combined with muscular rigidity and marked tenderness, is a most important sign of early peritonitis.

Extreme retraction of the abdomen occurs in wasting of the subcutaneous and omental fat and atrophy of the abdominal organs.

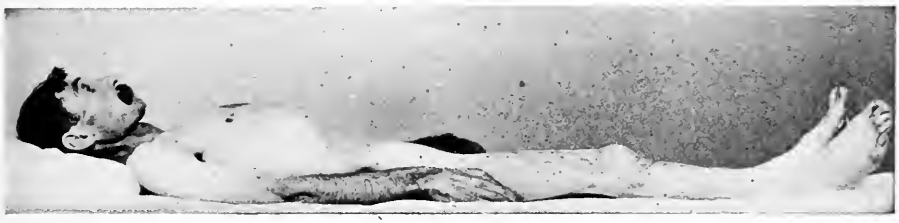


FIG. 37.—Scaphoid abdomen caused by starvation in a case of œsophageal carcinoma.—Jefferson Hospital.

Local retractions of the abdomen are not of importance as physical signs. They are seen around the base of large hernias, especially in the lateral regions of the abdomen, in large ventral hernias, and in the upper regions in diaphragmatic hernias. These areas of depression disappear when the hernias which cause them are reduced.

In moderately large peritoneal effusions of some standing, when the patient assumes the lateral decubitus the side of the abdomen which is uppermost shows a concave retraction while the anterior and dependent portions bulge more prominently.

General Distention of the Abdomen.—This condition may be caused by subcutaneous and intra-abdominal fat, the excessive accumulation of gas in the stomach or intestines, fluid in the abdominal wall or peritoneal cavity or both combined, or a large intra-abdominal tumor or cyst.

Subcutaneous and intra-abdominal fat accumulations in the obese frequently cause enormous distention of the belly. In such cases there is excessive and often irregular development of the panniculus adiposus elsewhere; while in ascites and tumor the general nutrition is usually impaired. In cases where there is reason to suspect pregnancy or the presence of an abdominal tumor a large deposit of fat may render the diagnosis difficult. Large accumulations of fat in the omentum, such as sometimes occur in persons of middle age, may simulate pregnancy or a tumor. Fat in the belly walls interferes greatly with the examination by means of the X-rays.

The Excessive Accumulation of Gas — Meteorism, Tympanites. — The distention is symmetrical and may be extreme. There is tympanitic percussion resonance and absence of fluctuation. The association of these physical signs renders the diagnosis easy. When extreme the condition causes restriction of respiratory movement, the disappearance of the respiratory excursus in the epigastric zone, and displacement of the cardiac impulse upward as high as the fourth interspace and to the left of its normal position.

Moderate distention may result from injudicious eating, acute and chronic gastro-intestinal disorders, especially in neurotic persons, and the slight paresis of the intestines which occurs in acute febrile diseases, as enteric fever or pneumonia. Nervous women are apt to "bloat," as it is popularly called, after eating. Excessive tympany occurs in grave cases of the infectious diseases, as enteric fever with deep ulceration, septic conditions, acute general peritonitis, intestinal obstruction, after the release of a constricted loop of intestine after operation, as in strangulated hernia, and in some cases of hysteria.

FIG. 38.—Ascites, caused by cirrhosis of the liver.—Jefferson Hospital.

Free gas in the peritoneal cavity occurs as the result of the perforation of an air-containing viscus into that space. The abdomen is greatly and uniformly distended, its surface tense and smooth, the outlines of intestinal convolutions and vermicular movements are not visible, and the respiratory movement of the upper part of the abdomen ceases. The most common causal conditions are peptic ulcer of the stomach or duodenum, a perforating typhoid ulcer, and ulcerative or necrotic appendicitis. As the air occupies the highest region of the cavity, it causes a disappearance of the normal percussion dulness of the liver and spleen, which is replaced by a tympanitic note in these areas, of the same character as that over the abdomen elsewhere. The mere disappearance of the hepatic dulness does not, however, justify the diagnosis of pneumoperitoneum, since the intestines and especially the transverse colon may occupy the space between the liver and the wall of the thorax and separate them completely. Moreover, the liver dulness may be greatly diminished in pulmonary emphysema of high grade, atrophic cirrhosis of the liver and acute yellow atrophy. The diagnosis of free air in the peritoneum may, however, be determined by careful percussion in the axillary line according to the following procedure: In the dorsal posture there is dulness alike in the condition under consideration and when the liver is separated from the wall of the thorax anteriorly by the distended intestine. When, however, the patient is turned upon his

left side there always remains a limited area of dulness in the axillary line high up in the case of meteorism, while the dulness wholly disappears in the case of pneumoperitoneum. The same method of examination is applicable to the spleen, although the small size of this organ renders its recognition alike in large meteorism and in pneumoperitoneum a matter of much greater difficulty than in the case of the liver.

Fig. 39.—Ascites.—German Hospital.

Fluid in the Abdominal Wall or Peritoneal Cavity.—An excessive dropsy of the wall in some cases of anasarca may cause distention of the abdomen. This condition is encountered in acute nephritis and in the later stages of cardiovascular disease. The abdomen is tense, doughy, and pits upon pressure; the more dependent parts of the body, feet, ankles, legs, thighs, and pudenda, are highly œdematous, and the condition is usually associated with effusion into the peritoneum and sometimes also into the other serous cavities. The pallid and puffy facies in acute nephritis is characteristic.

Ascites or free fluid in the cavity of the peritoneum yields characteristic physical signs. The enlargement of the abdomen is general and symmetrical. Its degree and outline depend upon the amount of the fluid and the fact that under the influence of gravity it changes its position with changes in the posture of the patient. In moderate effusions, in the dorsal decubitus the middle of the abdomen is more or less flattened while the lateral regions bulge outward, in the lateral decubitus the lower lateral and anterior walls of the

Fig. 40.—Pregnancy—ninth month.

belly protrude while that which is uppermost is slightly incurved, in the knee-elbow posture the weight of the fluid causes the abdomen to sag down in an unusual manner, and in the erect posture the lower segment of the abdomen is especially prominent. In all these positions there is dulness upon percussion over the dependent areas and tympanitic resonance

over the upper, since the fluid gravitates toward the dependent regions of the cavity and the air-containing intestines float upon it in the upper spaces. Ascites in a belly previously relaxed or pendulous causes in the erect posture a prominent and somewhat conical symmetrical protrusion of the lower parts. Massive ascites gives rise to uniform symmetrical enlargement of the abdomen, but little influenced by change of posture.

Fig. 41.—Dilatation of colon. Male, 12 years old.—Rotch.

Ascites results from pathological processes directly implicating the peritoneum, as ordinary infections or tuberculous inflammation or cancer, or the portal vessels, as the pressure of new growths, gall-stones, cancerous invasion, extreme sclerosis or pylephlebitis due to other causes, or disease of the liver. Cirrhosis of the liver is a common cause of ascites. Tumors of the abdomen and especially large solid tumors of the ovary are frequently attended by ascites. The foregoing have been spoken of as local causes. The general causes of ascites are those which give rise to anasarca and effusion into the other serous sacs. Peritoneal effusion resulting from local causes is not usually at first associated with œdema of the lower extremities. As the fluid accumulates it exerts pressure upon the large abdominal veins, especially the iliacs and ascending vena cava, giving rise to dropsy.

Tumor as a Cause of General Abdominal Enlargement.—The pregnant uterus, ovarian, pancreatic, and hydatid cysts, and large new growths cause distention which may simulate that due to the causes just considered. The enlargement caused by these conditions differs from that caused by fat, tympany, or fluid in being usually more prominent in the anteroposterior than in the bilateral diameter, not so symmetrical, and not yielding uniform signs upon palpation and percussion. Other causes of general enlargement of the abdomen are fecal accumulation, cancer of the bowel, disseminated cancer of the peritoneum, and large peritoneal or retroperitoneal sarcomata and lipomata. To this list must be added hydronephrosis and enormous dilatation of the stomach or colon.

Local Prominence of the Abdomen.—Circumscribed swellings or tumefaction may be caused by abnormal conditions of the belly wall or of the contents of the cavity. These changes in contour should be carefully sought for in all cases presenting symptoms referable to the abdominal viscera. The methods of especial value are inspection, palpation, and percussion. In thin persons radioscopy yields important results.

The recognition of the nature of local bulgings in the abdominal wall is as a rule not attended by great difficulty, but the diagnosis of visceral

tumors is frequently obscure and in many cases can only be positively determined by an exploratory operation.

Local Prominences due to Changes in the Wall of the Abdomen.—These comprise abnormal conditions of the muscles, irregular collections of subcutaneous fat, hernia, abscess, enlarged lymph-glands, and neoplasms, particularly sarcomata.

A spasmodically contracted rectus muscle may simulate a tumor. The diagnostician must be on his guard against the appearance and sensation imparted to the touch by a contracted right rectus in the pyloric region.

Phantom Tumor.—The condition known as phantom tumor, due to persistent gaseous distention of a knuckle of gut with spasmodic contraction of the overlying muscle, causes a tumor-like swelling. Such swellings

Fig. 42.—*a*, epigastric hernia; *b*, hernia reduced.

appear and disappear, with alterations in contour and position; sometimes subside under gentle friction with the warmed hand and always under anæsthesia. They occur in hysterical persons. Fitz has suggested that in some of the cases phantom tumors are symptomatic of congenital or acquired dilatation of the colon.

Fat. — In very obese persons remarkable rolls and masses of subcutaneous fat collect in the abdominal wall. These are usually but not always symmetrical in arrangement, and may simulate tumors, from which they may be differentiated by their continuity with the panniculus adiposus, their consistency and want of tenderness, and the general condition of the patient. Circumscribed fatty tumors—lipomata—are common. They are hemispherical or egg-shaped, elastic, painless, somewhat movable, and more common in the lateral and posterior aspects of the trunk than in the abdominal wall. They frequently occur in spare persons.

Hernia.—No examination of the abdomen is complete that does not include the sites of hernia. This is especially important in cases attended

by intestinal obstruction and vomiting. or persistent pain in the inguinal region. The inguinal and femoral regions should be examined by palpation under the cover of the sheet or clothing and if necessary by inspection as well. Ventral and umbilical hernias and scar-hernias after operation may be readily recognized. The tumor varies in consistency according as it consists wholly of gut or partly of omentum. It is usually soft, without pain upon manipulation, and reducible. It varies in size from a mere nodule to a sac containing a large portion of the abdominal contents. It very often disappears spontaneously when the patient assumes the recumbent posture. or is then readily reduced. Strangulated hernia does not, as a rule, yield to taxis.

Abscess.—Purulent collections in the abdominal wall may be recognized by the signs of inflammation, swelling, redness, heat, and pain, by their contour, and especially by fluctuation. Pus may form in any part of the wall or find its way to any point upon the surface. Appendiceal abscess usually forms a circumscribed, fluctuating tumor in the right lower quadrant of the abdomen.

Lymph-nodes.—The superficial lymphatic glands of the groin do not form visible tumors unless distinctly enlarged. They are frequently palpable as small nodular bodies in adults who are in good health. They may become enlarged and tender in injuries of the leg or foot, in venereal disease, and in common with the superficial lymph nodules in other parts of the body in some of the acute infectious diseases and in particular in the bubonic plague. Slight enlargement of the inguinal lymphatics is common in generalized malignant diseases—carcinomatosis, sarcomatosis. Massive enlargement of these structures takes place in Hodgkin's disease. The enlarged inguinal glands in venereal disease and the plague frequently form suppurating buboes.

Neoplasms of various kinds may develop in the abdominal wall. The most common variety is sarcoma. In sarcomatosis cutis many small subcutaneous nodules appear scattered over the abdomen. In a recent case a sarcoma developed at the umbilicus and was followed, after operation, in about a year by a small nodule in the immediate neighborhood and many others in different parts of the body.

Local Prominences due to Abnormal Conditions within the Abdominal Cavity.—These conditions comprise:

Temporary Dilatation of the Stomach from Excesses at Table,
Gastrectasis,
Local Gaseous Distention of the Bowel, and Intussusception.
Fecal Accumulations,
Ectopic or Floating Viscera,
Visceral Hypertrophies and Enlargements,
Intra- and Perivisceral Abscess,
Abscess from Caries of the Spine,
Cysts,
Extra-uterine Pregnancy,
Abdominal Aneurism,
Glandular Enlargements, and
Malignant and other New Growths.

Any of these can exist without being the occasion of prominence recognizable upon inspection; but under favorable conditions this method of physical examination yields suggestive—even positive—physical signs. It is usually a question of the degree of their development respectively.

Excessive fat and muscular rigidity mask the signs of these conditions, and in the case of great abdominal tenderness a satisfactory examination is impossible. The plain recognition of an abdominal tumor does not in all instances justify a further diagnosis of its cause or nature.

Gastrectasis.—Temporary dilatation of the stomach from excesses in eating causes in persons who are not obese a visible prominence in the epigastric region. Substantive gastrectasis from any cause shows a bulging of the abdominal wall in the region of the umbilicus or above it. This bulging has downwards and to the left the outline of the greater curvature; if the stomach, as is very commonly the case, is displaced downward and its longitudinal axis more vertical than normal, the outline of the lesser curvature may also be visible below the ensiform cartilage.

Fig. 43.—Visceroptosis.— Pennsylvania Hospital.

Intestinal Obstruction.—The entire abdomen may be distended or only parts of it. If the colon be distended by hard fecal masses the course of the bowel is marked by an elongated eminence, the contours of which correspond to those of the gut. If the intestinal stenosis be acute a local area of gaseous distention without peristalsis occurs above the obstruction. The obstruction may be caused by fecal accumulations, large gall-stones, enteroliths, or the pressure of a tumor. Any of these may give rise to a distinct, localized, asymmetrical prominence of the abdominal wall.

Intussusception is most common in childhood and shows itself as an elongated sausage-shaped tumor usually in the region of the cæcum or at the sigmoid flexure.

Ectopic or Floating Viscera.—General splanchnoptosis—Glénard's disease—causes a prominence or protrusion of the lower segment of the abdomen and is common in women, being favored by tight corsets, the method of supporting the skirts, and the relaxation due to childbearing. Enteroptosis causes a similar deformity more or less marked; gastroptosis is usually associated with dilatation of the stomach. A vertical position of the stomach may be congenital or acquired as the result of tight lacing. The pylorus then occupies a position in the median line or to the left of it, and the greater curvature lies below the level of the umbilicus. In thin persons these displacements of the organ may sometimes be demonstrated by the methods of physical diagnosis, especially if it be inflated with gas followed by the introduction of water through the tube so that the greater curvature may be determined by dulness on percussion in sharp contrast with the tympanitic resonance of the colon.

Floating kidney sometimes gives rise to an oval prominence plainly visible upon inspection, which may be made to shift its position or dis-

appear upon manipulation or upon changes in the posture of the patient. The swelling caused by a displaced kidney is usually upon its own side, but when very movable it may sometimes be forced beyond the median line to the opposite side. It may occupy a position anywhere between the ribs and the pelvis and is freely movable with deep respiration. *Ren mobilis* is much more common in women and upon the right side.

Floating Spleen.—The normal spleen which, in consequence of elongation of the gastrosplenic ligament and the splenic artery and veins, has become dislocated—*lien mobilis*—does not cause a visible abdominal swelling. When, however, the displaced organ is also enlarged, as is frequently the case, there may sometimes be seen a rounded swelling upon the left side in any position from the hypochondrium to the pelvis. This swelling, like that caused by the dislocated left kidney, with which floating spleen is often associated, is freely movable upon manipulation and change of posture.

Floating liver is among the rarest of clinical or anatomical findings. The dislocation of the organ is usually slight. There is general enlargement of the right lateral region and a large mass of characteristic outline which descends when the patient assumes the erect posture. Tympany in the upper part of the right hypochondrium—normal area of liver dulness—disappearing when the organ is replaced, and the well-defined lower border of the liver upon palpation, render the diagnosis a matter of comparative ease.

Enlargement of the Gall-bladder. — This condition may properly be considered at this point, since the position of the enlarged bladder is very different from that of the normal gall-bladder. The enlargement is the result of cholecystitis, frequently associated with cholelithiasis, or carcinoma.

The gall-bladder is distended by a serous fluid which gradually accumulates in consequence of the inflammatory changes in its walls,—dropsy of the gall-bladder,—the bile no longer entering, because of obstruction of the cystic duct by a calculus, a plug of tenacious mucus, adhesive cholangitis, or a carcinomatous nodule. In comparatively rare instances infection by pyogenic organisms causes suppurative cholecystitis—empyema of the gall-bladder. If the gall-bladder be sufficiently distended and the abdominal wall thin, there may be seen an elongated, smooth prominence in the region of the notch of the liver, projecting below the liver margin and rising and falling with the respiratory movements. The gall-bladder may be greatly distended, reaching in some instances the size of the fist or more. It is then sometimes pear-shaped, the fundus being freely movable from side to side upon manipulation and change of posture.

In some instances when the cholecystitis is associated with cholelithiasis the gall-bladder is distended by an enormous accumulation of calculi; in others the tumor may be due to primary or secondary carcinoma of the gall-bladder.

Visceral Enlargements.—The so-called corset liver may give rise to a visible prominence in the right lateral region, reaching as low as the crest of the ilium and moving with respiration. In cases in which the pressure constriction is marked the portion of the liver below it is movable and may simulate, especially when a loop of intestine occupies the groove, a displaced kidney or new growth in the ascending colon.

Enlargement of the liver, causing marked prominence in the right hypochondrium, in some cases of the entire abdomen, may be due to hypertrophic cirrhosis, carcinoma, amyloid disease, conditions causing obstructive jaundice, leukæmia, syphilis, hyperæmia due to cardiac disease, and fatty liver.

Enlargement of the spleen may attain a considerable degree before it gives rise to signs upon inspection. Massive enlargement may occur in chronic malaria—ague cake, leukæmia and pseudoleukæmia. The organ may reach to the pelvis and even to the right of the median line.

Enlargement of the Kidneys.—Renal tumors develop from behind forward, tending to displace the movable organs of the abdomen, especially the intestines, aside.

The anatomical relations of the ascending and descending colon are such that these portions of the intestines, being attached to the kidneys by connective tissue, are retained in front of the growing renal tumor and tend to obscure its dulness upon percussion. The development of the tumor from the upper portion of the kidney causes a prominence of the hypochondrium on the corresponding side, which extends as the growth develops to the iliac region; the development of the tumor from the lower portion of the kidney causes prominence first in the iliac region. Two solid new growths of the kidney only are of clinical importance from the stand-point of diagnosis, namely, carcinoma and sarcoma. The former is more common in advanced life, the latter in childhood; the former is apt to cause early cachexia, while in the latter the general nutrition may be maintained; finally, sarcomatosis of the skin in connection with tumor of the kidney is highly suggestive as to the nature of the renal affection. Renal adenoma cannot be differentiated from carcinoma during the life of the patient. Much more rare is hypernephroma.

Fig. 44.—Massive enlargement of spleen in a case of splenomedullary leukæmia.—Jefferson Hospital.

In the rare cases in which both kidneys are involved the abdominal enlargement is of course bilateral.

The very rare primary malignant disease of the suprarenal capsules may give rise to a tumor in the hypochondrium of the corresponding side, which differs in no respect from the similar manifestation caused by a tumor of the upper half of the kidney. Renal tumors move only slightly or not at all with respiration.

Enlargement of the pancreas is caused by chronic pancreatitis and carcinoma. It very rarely reaches such a size as to occasion visible prominence in the epigastrium.

Abscess.—Local bulgings of the surface may be caused by suppurative inflammation in and around the abdominal viscera.

Abscess of the Liver.—Multiple abscess does not usually reveal itself by changes in the contour of the surface. Tropical abscess commonly causes the liver to enlarge upward, especially upon the right side. The respiratory excursus is diminished or absent and the lower intercostal spaces obliterated. There is often local œdema.

Subphrenic abscess occasions marked downward displacement of the liver and a smooth, soft tumor in the epigastrium. If, as is commonly the case, there is air as well as pus in the subphrenic space, the diagnosis is not attended with difficulty.

Abscess of the spleen, when of sufficient size, sometimes reveals itself by a splenic tumor, upon the surface of which a fluctuating area or areas may be obscurely felt through a thin-walled abdomen.

Renal abscess may cause a circumscribed tumor in the hypochondrium or iliac region of the affected side, with obscure fluctuation, but without œdematous swelling of the neighboring tissues.

Perinephritic Abscess.—The swelling occupies the lumbar region and there is œdema of overlying and adjacent parts. There is frequently burrowing of the pus in a downward direction, so that a second fluctuating tumor may be present at a more dependent point.

Abscess in Appendicitis.—The common situation of the large, circumscribed intraperitoneal abscess is in the iliac region between the navel and the anterior superior spine. The abscess may form in the retroperitoneal space and burrow beneath the iliac fascia, showing itself at Poupart's ligament, or it may accumulate in the retroperitoneal tissue in the flank, forming a large paranephritic abscess, with the usual œdematous condition of the surrounding parts.

Abscess from caries may follow enteric fever and show itself as a small fluctuating tumor overlying a rib or costal cartilage. Vertebral caries may cause an abscess in the lumbar region, or the pus may follow the sheath of the psoas muscle and point below Poupart's ligament—psoas abscess.

Ovarian or tubal abscess may give rise to distention in the iliac region of either side. When upon the right side these conditions may simulate appendiceal abscess, with which they are also occasionally associated.

Cysts.—Local as well as general prominence may be caused by cysts of various kinds. If large the distention is general, if small it is local and circumscribed. From a pathologico-anatomical beginning wholly without symptoms and unrecognizable, certain cysts frequently attain enormous dimensions. Among these are especially to be mentioned cysts of the pancreas, hydronephrosis, and ovarian cysts which are often of such size as to simulate ascites. The smaller cysts do not present physical signs which differentiate them from abscesses in the same localities. It is only by a general knowledge of the pathological processes which give rise to cyst- and abscess-formation respectively and a careful consideration of the anamnesis and associated symptoms that the differential diagnosis

can in some instances be made out, as in dropsy and empyema of the gall-bladder, echinococcus and abscess of the spleen, or hydro- and pyonephrosis. Cysts springing from the liver, dropsy of the gall-bladder, echinococcus and pancreatic cysts have their early manifestations in the upper regions of the abdomen—epigastric zone—to the right and left of the median line respectively; those springing from the kidney—hydronephrosis, echinococcus—first appear in the lateral regions, while those from the pelvic organs, ovarian cysts, hydramnion, arise from the pelvis—hypogastrium. Mesenteric cysts are usually situated to the right of the umbilicus and below its level. Cysts connected with the liver and spleen are influenced by the respiratory movements; those connected with the pancreas only slightly or not at all, and those developing from the kidneys, ureters, and pelvic organs remain unaffected by the respiratory movements of the diaphragm.

Aneurism.—Aneurism of the abdominal aorta may cause a distinct, pulsating tumor commonly in the epigastrium but occasionally to the left of the median line in front or in the lumbar region. This tumor is almost always immovable, but in rare instances has been influenced by manipulation and change of posture, but not by respiration. It presents the signs of aneurism, and is to be differentiated from tumors overlying the aorta and from the so-called "dynamic pulsation" of the aorta which occurs in neurotic individuals. The distended urinary bladder in urethral stricture, impacted calculus, etc., gives rise to a distinct rounded oval tumor of the hypogastrium, which reaches in extreme cases well up towards the umbilicus. To a less extent the retention of the low fevers and comatose conditions gives rise to a similar prominence. In the latter case, the incontinence of retention—*stillicidium urinæ*—prevents extreme distention. The anamnesis, the oval outline of the tumor, its central and symmetrical situation, fluctuation, and its immediate disappearance upon catheterization render the diagnosis clear.

Extra-uterine Pregnancy. — There is a history of morning nausea, paroxysmal colicky pain with faintness, enlargement and hardness of the breasts, and chloasma uterinum, together with the presence of a prominence to the right or left of the median line above the brim of the pelvis. Very often rupture of the sac takes place before it has attained sufficient enlargement to be recognized by the methods of physical diagnosis. This accident is attended by collapse symptoms, and upon vaginal examination the uterus is found to be somewhat enlarged and displaced downward and to the opposite side.

Glandular Enlargements.—Enlargement of the retroperitoneal glands, usually sarcomatous—Lobstein's cancer—may cause a visible tumor in the epigastric or umbilical region, usually tense, immovable, and nodular; sometimes slightly movable and obscurely fluctuating and crossed by the colon, which may be recognized upon palpation or by its tympanitic resonance, to secure which artificial inflation may be necessary. Tuberculous mesenteric glands—tabes mesenterica—cause, especially in children, marked protrusion of the abdomen with tympany. The enlarged lymphatic glands may cause irregular local prominence in the region of the navel or in the right iliac fossa and may be recognized upon palpation.

Malignant and Other New Growths.—Malignant diseases of abdominal organs—carcinoma, sarcoma—are of chief, while benign affections, fibroma, lipoma, myxoma, adenoma, and gumma, are of subordinate interest from the stand-point of diagnosis. This difference is to be ascribed not only to the greater frequency of the former and their disastrous effects upon the health and ultimately upon the life of the patient, but also to the fact that at some time in their course the diagnosis becomes both practicable and obvious, while in the latter with less significant symptoms the diagnosis cannot be made out and the condition often remains unsuspected during the whole course of the patient's life and only assumes pathologico-anatomical interest when the case, death having resulted from an entirely different disease, at length comes to autopsy. It is of diagnostic importance that in visceral as well as in external cancer, secondary implication of adjacent and distant organs takes place with characteristic signs and that ultimately in many cases the superficial lymphatic glands become enlarged, nodules appear in the skin and elsewhere—general carcinomatosis, general sarcomatosis.

FIG. 45.—Sarcomatosis cutis,—primary tumor springing from a pigmented mole on the forehead; metastatic growths appeared in a few months after primary growth.—Jefferson Hospital.

Cancer of the Stomach.—The tumor can be seen in some cases, but is usually only to be recognized upon palpation. It most commonly occupies the region of the pylorus and may be slightly movable with respiration and freely so upon manipulation. A visible tumor occupying the greater part of the epigastrium and even extending beyond its borders, irregular, nodular, well defined at its margin, immovable and very distinct through the emaciated wall of the belly, is sometimes present in advanced cases of carcinoma extensively involving the anterior wall of the stomach.

Cancer of the Liver.—The volume of the organ is usually greatly increased. The increase is rapid and may assume enormous dimensions. It may affect the entire liver or the right or left lobe to a preponderating extent. When the right lobe is chiefly involved, there is a flaring out of the lower ribs and costal cartilages; when the left, the appearance of the tumor may suggest a new growth involving the greater curvature of the stomach, a cyst of the pancreas, or an enlarged spleen, but these doubts are immediately set at rest by palpation and percussion. The surface is usually uneven and the border irregular, and these signs may in some cases be clearly made out upon inspection. In the absence of adhesions the respira-

tory excursus of the liver may be seen, and in one remarkable case in my service at the Philadelphia Hospital, when upon autopsy the entire right lung was found to be solidified by secondary carcinomatous infiltration, the respiratory movement of the liver, plainly seen through the abdominal wall, was from left to right upon inspiration. When, as is often the case, extensive adhesions are present, respiratory movement of the enlarged liver does not take place Multilocular echinococcus and gumma of the liver present great difficulties in diagnosis. Splenic enlargement in the former and a history of lues in the latter are significant. An individual who has syphilis may also be the subject of echinococcus disease.

Cancer of the Gall-bladder.—The position of the tumor and its respiratory movement are important. It is apt to be mistaken for cancer of the pylorus or duodenum. In the latter affections, when the cancer is primary, free hydrochloric acid may be wanting in the gastric contents, secondary dilatation of the stomach shortly appears, and the tumor may be made out to be connected with the stomach or bowel by dilating the stomach, with simultaneous percussion and palpation, while the seat of the tumor in the gall-bladder becomes at the same time more obvious by its shape and relatively superficial situation.

A tumor formed by cancer of the head of the pancreas cannot often be positively differentiated from cancer of the pylorus, duodenum, transverse colon, or porta hepatis. At best in a majority of cases the diagnosis must be made by exclusion.

Inspection of the Surface of the Abdomen. Abnormal Signs. — Moderate ascites and large tumors may be present without changes in the integument; but excessive distention causes nutritive changes and the skin loses its natural appearance, becoming tense, glistening, and thinned. White lines or striæ—*lineæ albicantes*—irregularly parallel and slightly depressed below the adjacent surface are produced by extreme or prolonged distention, as in pregnancy, obesity, and ascites. They are seen upon the abdomen, flanks, and thighs, and persist after the condition which caused them has passed away. Jaundice is often more conspicuous here than on surfaces exposed to the air. Striking deposits of pigment occur in the linea alba in pregnancy, especially in brunettes, and pigmentation due to abdominal growths and diseases of the peritoneum. Addison's disease, melanotic cancer, exophthalmic goitre, scleroderma, arteriosclerosis, and chronic heart disease is often conspicuous upon the abdomen, especially in the lower quadrants and about the flexures of the thighs. The pigmentation of vagabondage due to lice and filth is usually characterized by the parallel linear superficial lesions of scratching. The hæmochromatosis of hypertrophic cirrhosis and diabetes and in rare instances scleroderma are attended by conspicuous pigmentation. The prolonged administration of arsenic frequently causes marked discoloration of the skin. The general discoloration of argyria is less pronounced upon the surface of the trunk than upon the face and extremities. The specific eruptions of the exanthemata, especially the initial rashes of variola, and the rose spots of enteric fever are to be sought for upon the abdomen. Taches bleuâtres, tinea versicolor, and the symmetrical diffuse macular eruption of secondary syphilis are to be seen. The scars of surgical operations, especially those

performed for the relief of appendicitis. gastric and gall-bladder disease, and various diseases of the pelvic organs are common nowadays and may shed light upon many abdominal disorders—adhesions and the like—post-operative neurasthenia and other obscure maladies. Enlarged inguinal glands and retracted cicatrices in the groins may be significant of venereal infection.

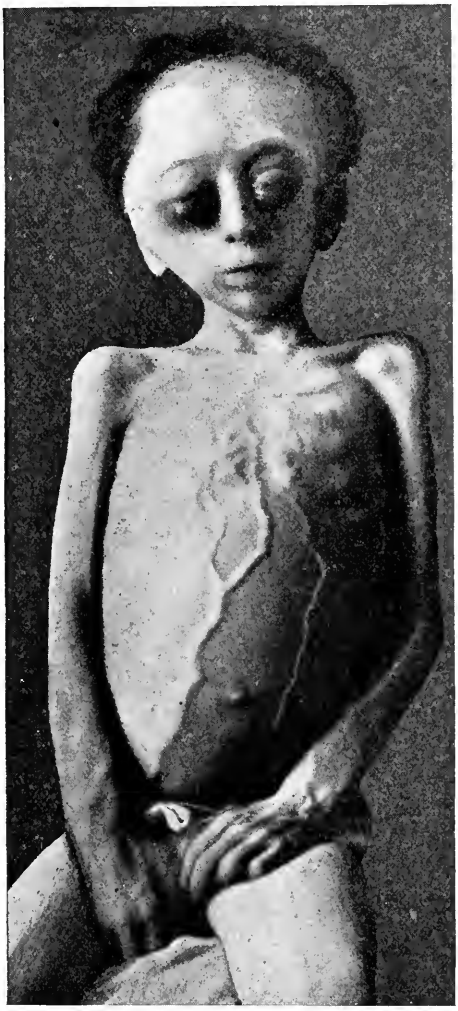


FIG. 46.—Sarcoma of spine, showing venous stasis and metastatic growth in orbit.—Young.

Vascular Changes.—Signs relating to circulatory derangements are enlarged superficial epigastric arteries and enlarged superficial veins. The former are exceedingly rare and indicate obstruction of the aorta or iliac arteries; the latter very common and constitute the evidence of collateral venous circulation in obstruction of the portal system or the inferior or superior vena cava. Among the common causes of such obstruction are, in the portal circulation, cirrhosis of the liver and tumor; in the general circulation, abdominal and mediastinal tumor, dilatation of the stomach of high grade, and ascites of long standing.

Caput medusæ is a varicose arrangement of the dermal veins around the umbilicus with radiating branches. It is made up by the dilated branches of the epigastric veins at their juncture with a large single vein which passes from the hilum of the liver and follows the course of the round ligament—para-umbilical vein of Sappey. Much more commonly the enlarged collateral veins are distributed irregularly over the surface of the abdomen and indicate one of the courses towards the right heart taken by the blood in pressure upon the inferior vena cava. There is engorgement of the blood from the lower extremities in the inferior epigastric and internal mammary veins, with dilatation of the superficial abdominal veins. In obstruction of the portal system and inferior vena cava the course of the blood in the dilated superficial veins is upward; when the superior cava is obstructed the course of the blood in the superficial veins of the chest and abdomen is downward, the blood seeking its way to the right heart

by means of the right azygos which communicates with various tributaries of the inferior vena cava. Pressure upon the innominate vein of the right or left side may give rise among other signs to great dilatation of the superficial veins of the thorax and abdominal wall.

The Umbilicus. — The navel normally shows transverse or slightly spiral folds of the skin and is moderately retracted. It is deeply so and funnel-shaped in obese persons and level with the surrounding surface or protruding in large ascites and pregnancy. It may be the seat of caput medusæ or hernia, inflammation or eczema, carcinoma secondary to gastric carcinoma or tuberculous infiltration secondary to tuberculous peritonitis. A mole in the region of the umbilicus may undergo sarcomatous changes.

Movements of the Abdomen in Disease. — Inspiratory retraction of the epigastrium is present in stenosis of the upper air-passages and imperfect action of the diaphragm. Diminished respiratory movement of the abdomen may be caused by upward pressure upon the diaphragm, as in tympany, ascites, and abdominal tumors on the one hand, or by massive pleural or pericardial effusions on the other. In the early stages of peritonitis abdominal respiratory movement is greatly impaired or wholly absent, on account of the pain and tonic contraction of the muscles of the wall; in the later stages on account of the tympany and upward pressure upon the diaphragm.

Visible Peristalsis.—In thin persons the normal peristaltic movements may sometimes be seen. They appear as wave-like, rounded elevations of the surface which may be attended by borborygmi and may be intensified by gentle irritation of the skin by the application of cold, brisk tapping or faradism. In some instances the peristaltic movements of the stomach from left to right are in sharp contrast to those of the transverse colon from right to left. In the wide separation of the recti occasionally seen in women who have borne many children these vermicular movements are very conspicuous.

The most important diagnostic significance of visible peristalsis relates to intestinal obstruction. The presence of peristalsis must be determined and whether or not it is always in the same direction and ceases at a certain spot. If the obstruction is at or above the ileocæcal valve the distended and mobile coils of small intestine occupy a position in the central portion of the abdomen, but if the obstruction involves the lower part of the large intestine—sigmoid flexure—the distention and movements of the bowel may be manifest in the region occupied by the ascending and transverse colon. The inflated fixed intestinal coil of acute stenosis of the gut, ileus—strangulated hernia—shows no peristaltic movement. Prior to immobility there is peristalsis. In chronic obstruction, after the muscularis of the gut has become hypertrophied, there is active peristalsis, with marked recurrent tumor subsiding with coarse borborygmi, just in advance of the stenosis. As the gas in the tumor is under tension, it does not yield tympanitic resonance but dulness upon percussion. Visible peristalsis in the left hypochondrium, with the vermicular contractions from left to right, has been observed in extreme gastrectasis.

Pulsation Synchronous with the Cardiac Systole.—Dynamic pulsation occurs in neurotic persons. It is seen in the median line and is often violent

but neither diffuse nor expansile. The pulsation of abdominal aneurism usually has both these characters and very often in addition systolic thrill and bruit. It is mostly situated in the median line, but may be seen in the left lateral region of the abdomen.

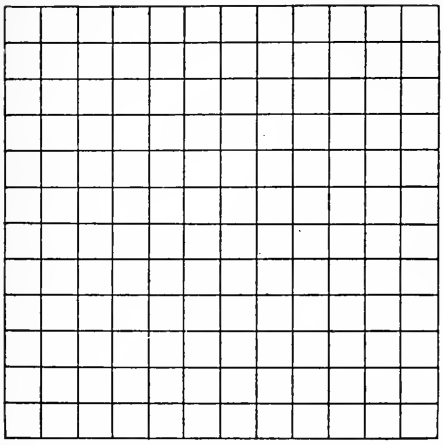


FIG. 47.—Beall's aid to inspection.

An Aid to Inspection in Circumscribed Movements not Well Defined.—I have found the following suggestion of K. H. Beall of much service:

"Over the area under inspection there is drawn with a skin pencil a square plaid figure, the squares of which are from 1.5 to 2.5 cm. in diameter and from 12 to 50 in number, according to the size of the area being studied. Any slight movement of the skin at any point in such a marked area causes a change in the direction of some of the lines and a distortion of the figure, and so renders visible movements of the internal organs which are not to be detected otherwise."

PALPATION.

The method of physical diagnosis in which the sense of touch is employed is known as palpation. It consists in the systematic examination of the surface of the chest and abdomen by the laying on of the hand. The physical signs elicited depend upon the condition and movements of the parts and the underlying structures. As in inspection, we study the form, size, condition of the surface, and movements. The method is applicable and essential to the examination of the thorax and abdomen.

Palpation in the Examination of the Thorax.

The chest should be bared, the attitude easy, the arms symmetrically disposed, the muscles relaxed. The examining hand should be warm and laid gently upon the surface. The amount of pressure employed must be determined in individual cases. Ticklishness, tenderness, and excessive fat constitute obstacles. The first may be overcome by care and diverting the attention of the patient; the others often amount to insuperable difficulties in the application of this method of diagnosis. The palmar surface of the whole hand is employed for a general survey, as in locating the position of the cardiac impulse or a thrill; the more sensitive finger tips for the study of the particular characters of such phenomena, for example the force and extent of the impulse or the coarseness or fineness and extent of a thrill.

By palpation we confirm and amplify the signs obtained by inspection, especially those dependent upon the form and contour of the chest,

the width of the interspaces, the presence of local swellings and deformities, and the respiratory and cardiac movements. These it is not necessary at this point to repeat. But there are other physical signs, not always recognizable upon inspection, which we investigate by palpation. These comprise the condition of the wall of the chest as regards

Muscular Tension,
Œdema,
Width of the Interspaces,
Fluctuation,
Nodes, Gummata, and Periosteal Thickening,
Location and Character of the Heart's Impulse.
Extracardial Pulsation and Diastolic Shock, and in particular the following physical signs which are exclusively within the scope of this method:
The Crepitation of Subcutaneous Emphysema,
Thrills, Cardiac and Vascular,
Fremitus, Vocal, Friction, and Rhonchal,
Tracheal Tugging.

Tension.—The tension of the muscular wall of the chest in the intercostal spaces and about the ensiform cartilage is not a sign of great value, yet it is to be studied in doubtful cases. The inspiratory retraction of the base of the chest is a sign of obstruction to the entrance of air, which may be at the larynx, as in œdema of the glottis, or in the smallest bronchial tubes, as in bronchopneumonia. The slight normal furrow of the lower intercostal spaces may be obliterated by pleural or pericardial effusion or a rapidly growing new growth. In old empyemata there is great relaxation and bulging and the cardiac pulsations may even be transmitted to the surface—pulsating empyema. Epigastric rigidity and tenderness are conspicuous in tetanus, and these phenomena are early symptoms in peritonitis beginning in the upper part of the abdomen.

Œdema.—Local œdema may indicate intrathoracic suppuration as in empyema or hepatic abscess, inflammation of the wall of the chest as in carbuncle, or obstruction to the venous circulation as in mediastinal tumor or aneurism. The puffiness involves the head and neck on both sides when the pressure involves the precava and is unilateral when it affects the right or left innominate only.

Spaces.—The width of the intercostal spaces may be felt when not seen upon inspection, and should be carefully investigated in cases of pleural effusion, since they are wide when the chest is distended and become narrow as the fluid undergoes resorption.

Fluctuation.—Elasticity or fluctuation in any prominence or tumor upon the surface of the chest is an important sign. It may be due to abscess of the wall itself, empyema necessitatis, cyst formation, or sarcoma. The differential diagnosis rests upon the associated clinical phenomena. In abscess of the wall the volume of the tumor is not affected by the respiratory movements; in empyema necessitatis the tumor diminishes upon inspiration and increases with expiration and the physical signs of intra-

pleural effusion are present; a cyst is usually sharply circumscribed, distinctly globular, tense, sometimes translucent, and commonly movable within a limited range.

Nodes.—Nodes upon the ribs, cartilages, or sternum or thickening at the chondrocostal or sternoclavicular articulations and periosteal thickening are important signs of disease. They may sometimes be felt when not obvious upon inspection, and their size and consistence can be recognized upon palpation. Among the earliest of the skeletal lesions of rickets is a nodular enlargement of the ribs at the juncture of the bone with the cartilage. These nodules are present upon the ribs of both sides and are symmetrical in their arrangement—the so-called rosary of rickets. Gummata are common upon the sternum and roughening and enlargement of the clavicles may be a manifestation of late syphilis. The clavicles are enlarged and the sternum deformed in acromegaly. Acute painful enlargement of the sternoclavicular articulation is not rare in gonorrhœal arthritis. A soft, elastic, slightly fluctuating tumor upon the upper part of the sternum may be a tuberculous abscess. Tender points are found upon palpation. They are not physical signs, but may be mentioned in this connection as symptoms of great value. They are found in intercostal neuralgia and correspond to the points of emergence of the intercostal nerves; in neurasthenia tender points are also found along the dorsal spine and the tenderness is very often present upon light and absent upon firm pressure; in necrosis of a rib; in fibrinous pleurisy and especially in that form of pleurisy which occurs in pulmonary tuberculosis, where the tenderness is most common and most marked in the infraclavicular region.

Apex-beat.—The precise location and character of the impulse of the heart. The palm of the hand should be first laid over the precordia below the left nipple. The signs elicited by inspection are thus confirmed and amplified. We determine whether the rhythm of the heart is regular or irregular and, if irregular, whether the arrhythmia is in time or in force or both, that is, whether there are differences in the intervals between the ventricular contractions, or in the power with which the heart contracts or these are combined. We observe also in this way the general character of the heart's action, that is, feeble or strong; heaving powerfully so as to move the whole chest, as in great hypertrophy or the overaction of mental or physical excitement—palpitation; that it has the diffuse slap often encountered in dilatation of the right ventricle, the sharp tap of mitral stenosis or the slow, heaving, forcible impulse sometimes met with in aortic stenosis.

The more sensitive tips of the fingers are next brought into service. They are placed over the point of maximum impulse and moved in various directions. The apex of the heart as determined by finger-tip palpation and by percussion is usually two or three centimetres below and to the left of the point of maximum or visible impulse. It frequently happens that the impulse not recognized upon inspection may be felt and rarely that a visible impulse cannot be appreciated by the trained touch. These two methods must be used in all cases.

Inspection and palpation yield the most satisfactory results in the study of the size of the heart. The base of the organ is fixed and is as a

rule not greatly displaced even by the pressure of an aneurism or new growth. To fix the position of the apex is to determine the long axis of the heart and gain a fairly correct idea of its size. The data obtained by percussion are much less definite, partly because of inherent difficulties in recognizing the limits of dulness in the rounded body of the heart surrounded by resonant lung and partly because of the modifying effects of pleural adhesions or effusion, gastric dilatation or abdominal tympany. When the impulse cannot be located by inspection or palpation, we employ auscultation and consider the clinical impulse to be near the point at which the first sound is most distinctly heard.

The changes in the relation of the apex to the wall of the chest caused by changes in the posture of the patient have already been considered.

Extracardiac Pulsation. — Pulsation beyond the limits of the heart is frequently seen, but its precise location, extent, and character are best studied by the sense of touch. A heaving impulse at the root of the neck occurs in hypertrophy, especially that form associated with aortic insufficiency and in overaction from nervous causes. It occurs also in anæmia and large hemorrhages, in apoplexy, and rarely in the stage of onset of intense infections, as variola. It is a conspicuous phenomenon in exophthalmic goitre. In neurotic persons the pulsating dilated transverse aorta may in rare instances be felt in the sternal notch—dynamic pulsation. Aneurism of the innominate artery or of the transverse portion of the aortic arch may give rise to similar pulsation. Anomalies in the distribution of the subclavian or thyroid arteries may also give rise to pulsation in this region. In old pleural adhesions at the apex and in pulmonary tuberculosis subclavian pulsation is often marked and extended. Pulsation commonly to the right of the manubrium, sometimes to the left of it, occurs in aneurism of the thoracic aorta and may often be felt when it is not seen. The force and extent of the impulse in pulsating empyema are best estimated by palpation.

Epigastric Pulsation. — This phenomenon is generally regarded as the sign of hypertrophy of the right ventricle and this view is unquestionably in some cases correct. The hypertrophied and overacting right heart communicates its movements to the tissues at the tip of and below the ensiform cartilage. The retraction corresponds to time with the ventricular systole and is due to the negative pressure caused by the alteration in size and diminution in the volume of the ventricles at this moment of the heart's revolution. Epigastric pulsation has been observed in cases in which no hypertrophy of the right ventricle has been found post mortem. Liver pulsation is much more frequently palpable than visible, and the distinction between this condition and a liver jogged by an overacting heart may often be made by bimanual palpation, since a pulsating liver expands and contracts, a jogged liver merely moves. Bimanual palpation, one hand upon the upper dorsal spine and the other upon the manubrium, may detect the expansile pulsation of a deep-seated aortic aneurism which presents no external signs. Diastolic shock is an important physical sign of aneurism. The tips of the fingers upon the sac in case erosion of the chest wall has taken place, or upon the surface directly overlying the sac, may often detect a diastolic shock, sometimes of considerable force.

Crepitation.—In wounds and operations upon the neck and chest air may find its way into the subcutaneous tissues and give rise to crepitation upon palpation. In rare cases this condition may result from the rupture of dilated peripheral pulmonary vesicles in emphysema.

Succussion.—When both fluid and air are present in a large space with rigid walls, as in pneumohydrothorax or pneumopyothorax, a distinct vibration or impulse may be felt upon shaking the patient or causing him to suddenly twist his body. This phenomenon, which is accompanied by a splashing sound, constitutes the sign known as Hippocratic succussion.

The arterial pulse is studied by palpation. This subject will be fully considered in a later section.

Thrills.—The palpable vibrations of the surface transmitted from the interior of the heart or arteries are known as thrills. They are usually confined to limited areas and may be easily overlooked unless the surface is first searched with the palmar surface of the open hand. They may then be studied with the finger-tips. They can frequently be felt only upon the lightest pressure, wholly disappearing if the pressure be increased. The sensation has been compared to that communicated to the hand by the purring cat—*frémissement cataire*. Thrills are usually felt during a portion of the cardiac revolution only—presystolic, systolic, post-systolic. They may disappear when the heart is acting feebly and become manifest again when, with general improvement in the condition of the patient, the heart contracts with greater power. They usually correspond in the time of the cardiac cycle with audible murmurs or bruits and are significant of the same lesions and produced by the same mechanism, namely, fluid veins, the vibrations of which transmitted through tissues to the surface are realized by the ear as murmurs, by the touch as thrills. In other words the thrill is the sensory equivalent of the murmur. The fact that very coarse thrills, especially the presystolic thrill, sometimes occur when no murmur can be heard, does not militate against the foregoing statement, since regular vibrations may be palpable though not frequent enough to produce sound. It is in accordance with these statements that thrills vary in the rapidity of their vibrations—fineness, coarseness—and that the finer thrills correspond to the higher pitched murmurs and the reverse.

A thrill at the base of the heart of maximum intensity in the aortic area is common in aortic stenosis.

A thrill of coarse quality, limited in extent, presystolic in time, more marked during expiration, and most distinctly felt in the fourth or fifth intercostal space inside the midclavicular line—mitral area—is the sign of mitral stenosis.

A systolic thrill in the same area is sometimes present in mitral incompetence and in rare instances in aortic stenosis.

A thrill often accompanies the presystolic murmur of aortic incompetency—Flint's murmur.

A thrill diastolic in time is occasionally felt in aortic incompetency, but is not very common.

Thrills are common in congenital defects of the heart.

A thrill systolic in time at the second left costal cartilage and interspace—pulmonary area—is occasionally observed in exophthalmic goitre;

very rarely it is a sign of pulmonary stenosis. A diastolic thrill in this area may be the sign of a rare condition, pulmonary incompetency.

A thrill over the lower portion of the sternum and at its right border—tricuspid area—sometimes occurs in dilatation of the right ventricle and tricuspid incompetence.

Systolic thrills when beyond the limits of the precordial space are more likely to be indicative of thoracic aneurism than of valvular disease. They are oftenest felt to the right of the sternal border and above the fourth rib, but may be present in the left side in a corresponding region. They are more common in aortic dilatation than in sacculated aneurism.

It is important to recognize the difference between a thrill and the slight shuddering tremor which may be felt in the merely overacting heart, as in palpitation from any cause.

Fremitus.—*Fremere*, to roar or murmur as a crowd or mob; technically, palpable vibration. The difference between a thrill and fremitus is much more readily recognized than described. It is, however, an essential difference and depends upon the difference in the mechanism by which they are respectively produced. Fremitus is usually much coarser than thrills, the vibrations are irregular and variable, the extent is far wider, and fremitus, even when produced by the movement of the heart as in the friction fremitus of pericarditis, does not constantly conform to definite movements in the cardiac cycle. Fremitus is a tactile phenomenon communicated to the surface of the chest by the act of phonation—vocal fremitus; by the friction of roughened surfaces against each other—friction fremitus; or by the respiratory movement of exudates of varying consistency within the bronchial tubes—rhonchal fremitus.

Vocal Fremitus.—This physical sign is of great value in the diagnosis of diseases of the respiratory organs. It is frequently spoken of as tactile fremitus, but erroneously so, since all fremitus is tactile. The hand is laid upon the bared chest while the patient counts "one, two, three," or repeats some words, as "twenty-one" or "ninety-nine." Under normal circumstances the fremitus is more intense in men than in women, in adults than in children, and in persons whose voices are powerful and low-pitched than in those whose voices are feeble and shrill. The patient should be asked to repeat the same phrase as the examining hand passes from one part of his chest to another, and to let his speaking be loud, low, and slow, always as nearly as possible in the same tone. This method of physical diagnosis is without value in persons suffering from aphonia or in those so feeble that they can only use the whispering voice. It is practicable in young infants who cry during the examination.

Vocal Fremitus in Health. — The vibrations of the vocal cords in phonation are transmitted along the walls of the trachea and bronchi and the column of air which they contain to the surface of the chest, which is thus set into vibration from within. These vibrations vary in different regions normally, and are most distinct where the large bronchial tubes approach the chest wall, less distinct where the mass of intervening vesicular tissue is greatest, and feeble or absent where the lung tissue does not come into contact with the wall, as in the precordial space. Pathological conditions which increase the capacity of the lung to conduct vibrations,

as consolidation, intensify the vocal fremitus; those which separate the lung from the wall, as pleural effusions, diminish or abolish it, as the case may be. There is normally considerable difference in the intensity of the vocal fremitus in the two sides, especially in the upper regions. This inequality is to be constantly borne in mind. The vibrations are more intense on the right than on the left side, in the upper (subclavicular) region than in the lower (inframammary), and in front than behind. It is feeble over the scapulæ, and usually absent or very feeble in that portion of the precordial space which corresponds to the area of superficial cardiac dulness. A thick layer of subcutaneous fat impairs the value of this physical sign, while a thin, elastic chest wall and deep voice render it very useful.

Vocal Fremitus in Disease of the Respiratory Organs.—The vibrations are intensified by conditions which cause consolidation of the lung, as tuberculous infiltration, croupous and bronchopneumonia, hypostatic congestion and atelectasis; they are enfeebled or absent altogether in pathological conditions which separate the periphery of the lung from contact with the chest wall, such as pleural effusion, pneumothorax, and cysts or tumors in the pleural cavity. Pleural thickening is usually attended with enfeeblement of the vocal fremitus proportionate to its degree, and, as a much thickened pleura gives rise to impairment of resonance, the differential diagnosis between a moderate effusion and pleural thickening may be attended with difficulty.

Temporary disappearance of vocal fremitus in pneumonia in an area corresponding to a lobe or part of a lobe may be caused by the plugging of a large bronchus with a mass of tenacious mucus. In the same manner a foreign body may cause localized absence of this sign. In infants and less frequently in adults distinct vocal fremitus is occasionally encountered upon the affected side in large effusions—a very puzzling phenomenon. The most probable explanation of this anomaly in children is that the intense fremitus caused by violent crying is transmitted along the elastic chest walls from the sound to the affected side of the chest; in adults, that tensely stretched strands or bands, the result of partial adhesions caused by a former attack of fibrinous pleurisy, conduct the vibrations from the compressed lung to the wall of the chest. In a moderate effusion under favorable circumstances the following variations may be recognized: normal vocal fremitus over the apex, enfeebled fremitus in the mammary region, and the complete absence of this sign at the base.

If the limitations of its usefulness be borne in mind, vocal fremitus is a sign of very great value, but it may mislead the unwary. In massive pericardial effusion it is of great service in the differential diagnosis between that condition and large left-sided pleural effusion.

Friction Fremitus.—In health the smooth and moist pleural and pericardial surfaces move upon each other without appreciable sound. When these surfaces are the seat of a fibrinous exudate they cause friction sounds which vary with the arrangement and density of the exudate and the energy of the respiratory or cardiac movements as the case may be. The vibrations which cause the sounds are transmitted to the surface and constitute the tactile sign known as friction fremitus. The sensation communicated to the examining finger is that of grating or rubbing and varies

from the finest grazing to a coarse attrition. It corresponds in location and extent with the friction sound which is its auditory equivalent.

Pleural fremitus is common in the infra-axillary region or below the nipple and is not transmitted beyond a limited area.

Pericardial fremitus, which is the sign of fibrinous or dry pericarditis, is felt in the precordial space over the right ventricle. It does not usually correspond in time accurately with the systole or diastole, gives the impression of being very superficial and is limited to a circumscribed area. It differs from the thrills felt over the heart in the tactile qualities referred to in a preceding paragraph.

Pleural fremitus and pericardial friction fremitus disappear as effusion takes place, separating the roughened surfaces, and as adhesions develop, by which the surfaces are united.

Rhonchal Fremitus. — Coarse bronchial râles, both dry and moist, sometimes communicate irregular vibrations to the surface of the chest readily recognized upon palpation. This form of fremitus is common in young children and may occur in thin-chested adults. It differs from friction fremitus in being coarse and more irregular and varying in intensity and quality with the râles that cause it. The sign is of little diagnostic value.

Tracheal tugging, a sign first described by Oliver, is of great value in the diagnosis of deep-seated thoracic aneurism. "Place the patient in the erect position and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb and use steady and gentle upward pressure on it, when, if dilatation or aneurism exists, the pulsation of the aorta will be distinctly felt transmitted through the trachea to the hand." A better method consists in the application of the index and middle fingers of the same hand on the sides of the cricoid cartilage, or the physician may stand behind the patient, who is seated, and place the forefingers upon the sides of the cricoid, with gentle upward pressure. The downward tug may be readily recognized. The tug is due to the fact that the arch of the aorta passes over the left primary bronchus in such a manner that when the aorta is dilated it impinges upon the bronchus with each pulsation. The tension of the bronchus is communicated through the trachea to the larynx. A downward tug felt only upon inspiration is frequently present in health and has no diagnostic value. Pulsation transmitted from the vessels of the neck to the cricoid must not be confounded with tracheal tugging. The movement of the former is forward and backward; of the latter a distinct downward pull with release.

Palpation in the Examination of the Abdomen.

This is the most valuable of the methods of physical diagnosis in diseases of the organs below the midriff. The patient should be in bed and the belly should be bared as for inspection. The hand of the physician should be warmed and applied to the surface with gentle pressure. One or both hands may be necessary. Bimanual palpation may be from side to side, the wall of the abdomen being deeply folded between the hands, or any accessible organ or tumor being thus investigated, or the

bimanual method may be used in the study of the lateral regions of the abdomen, one hand being placed in the lumbar region, the other in front. In this manner the border of the liver may be raised up against the anterior wall or a floating kidney thrust forward for examination, or deep fluctuation elicited in paranephritic or appendiceal abscess, or a hydronephrosis studied, or the contour of an enlarged spleen or carcinoma of the sigmoid flexure made out. When the object of the examination is to localize and determine the degree of tenderness it is better to study the face of the patient than to depend upon his statements or exclamations. It is also important to distinguish between superficial tenderness, as in cutaneous hyperæsthesia and the deep tenderness of an inflamed or tumid organ. It will frequently be found that here as elsewhere, in neurotic persons, more vivid expressions of pain are called forth by a light touch than by firmer pressure—a fact in itself of great diagnostic importance.

Excessive abdominal fat, muscular tension, and ticklishness are obstacles. The first often nullifies the results of palpation; the others may be overcome. Muscular tension due to apprehension, the excitement of the occasion, or other nervous causes may be overcome by elevating the head upon pillows and causing the patient to flex his thighs and knees; continuous deep or rapid breathing is also useful. Tact and address on the part of the physician and suggestion are also to be employed. It is frequently necessary to examine the patient under general anæsthesia before expressing a final opinion as to the nature of the case, and finally, there are serious cases of abdominal disease in which it may become necessary to perform an exploratory operation to arrive at a positive diagnosis. Ticklishness is an obstacle of minor importance, but it may call for the exercise of much patience on the part of both the doctor and the patient.

It often becomes necessary to turn the patient from side to side or to examine him in the knee-elbow posture, or standing. A digital examination by the rectum or vagina with or without bimanual manipulation is frequently required in lesions of the lower portions of the abdomen.

The regions of the abdomen must be in turn systematically explored, the natural rings and accidental sites of hernia examined, and the general outline, contour, and condition of the belly, particularly as to its symmetry and elasticity, carefully determined. Large knowledge of the changes caused by abdominal disease and wide experience are required in this field of diagnosis. Here also a delicate and well-trained touch—*tactus eruditus*—is especially serviceable.

The signs obtained by inspection are confirmed by palpation. Much knowledge is obtained by the latter method. This comprises the following subjects:

The Condition of the Abdominal Walls,
General and Local Fluctuation,
Pulsation, Thrill and Fremitus,
The Respiratory, Postural, and Manipulative Movements of Organs or Tumors,
Peristaltic and Fetal Movements,
The Outline and Relations of Palpable Tumors,
Their Density and Elasticity,
The Nature of the Surface of Tumors.

The Abdominal Walls.—In healthy young persons the belly walls are soft and elastic but neither tense nor relaxed, and the curvature of the abdomen as determined by inspection and palpation is symmetrical and uniform.

Abnormal firmness and relaxation are attended by a loss of healthy elasticity. Local firmness may be caused by inflammatory or carcinomatous infiltration, and general hardness by the massive enlargement of the liver, spleen, uterus, an ovary, or other organ, or diffuse malignant deposits in the intestines or peritoneum. Muscular rigidity is characteristic of the early stage of peritonitis. It may be localized, as in the right lower quadrant in appendicitis or enteric fever, or general. Local rigidity of the bellies of the recti is sometimes observed in neurotic persons and may be mistaken for a tumor, as a thickened or carcinomatous pylorus. Local rigidity with meteorism constitutes phantom tumor. A generalized inelastic doughy sensation upon palpation is often observed in tuberculous peritonitis. The general distention of ascites is associated with dulness save in the upper portions, where there is tympany, and with fluctuation; that of meteorism is associated with tympany everywhere, including the dependent parts, and a balloon-like elasticity quite different from that of the normal abdomen. The anasarcous abdominal wall is doughy, inelastic, and pits upon pressure; dropsical accumulations are seen in the flank and elsewhere in the more dependent parts.

Relaxation follows the resorption of large amounts of fat and repeated childbearing. In such cases the belly wall is often pendulous and remarkably puckered and thrown into folds when the patient lies upon her back. Relaxation also follows ascites of long standing and the removal of large tumors and is usually present in old age and the advanced stages of wasting diseases. In women who have borne many children wide separation of the recti is occasionally seen, the connective tissue of the linea alba being enormously stretched and thinned and the gastric and intestinal peristalsis plainly seen and felt over a large area in the middle of the abdomen. In such cases very large ventral hernia and downward displacement of the abdominal viscera—Glénard's disease—are commonly present.

Local tumors of the abdominal walls are abscess, attended by local induration and central softening; cysts, oval or circular in outline, tense, elastic and fluctuating; enlarged lymph-nodes in the inguinal region; subcutaneous carcinomatous and sarcomatous tumors, which may be movable or immovable, and arranged in irregular masses as is common in the former, or scattered singly over a wide area as in sarcoma; and hernia. The last appears in definite locations, as the inguinal and crural rings, the umbilicus, in the linea alba—*ventral hernia*—and in the sites of scars after surgical operations. Upon palpation the hernial tumor is usually soft, elastic and reducible; omental hernias are doughy and irregular in outline. The hernia which cannot be returned to the abdomen by manipulation is irreducible, that which is tightly constricted and is therefore likely to become or has already become sphacelated is strangulated.

The umbilicus that pouts in ascites or pregnancy is smooth, stretched, and somewhat translucent. In umbilical hernia the ring is usually distinctly felt; when omental the tumor at the navel is often large, firm,

irregular in its surface and irreducible and may suggest a malignant growth. The umbilicus, normally somewhat movable, when the seat of secondary carcinoma, usually by extension from the liver, becomes fixed and is indurated and nodular. Tuberculous infiltration of the tissues around the navel has been observed in tuberculosis of the peritoneum. A deeply seated, painful swelling of the navel is usually an abscess.

Fluctuation.—This sign is elicited by combined bimanual percussion and palpation, those methods being employed at the limits of the area examined, as for example at the right and left lateral regions of the abdomen in suspected ascites and at the opposite borders of circumscribed collections of fluid as in pancreatic or other cysts. To elicit general fluctuation the palpating left hand or finger-tips are lightly laid upon the surface of the right side of the patient's abdomen, while with the fingers of his right hand the examiner percusses or taps somewhat sharply upon the left side of the abdomen. If there be ascites a transmitted wave corresponding to each tap is felt upon the opposite side. This wave is also in many cases visible. Very light percussion may bring out this physical sign when the wall of the abdomen is thin. The thin ulnar border of the hand of an assistant must be rather firmly pressed against the abdomen in the middle line to arrest the undulatory transverse movement of the wall, which very often simulates the fluctuation of peritoneal effusion. This sign does not arise unless the fluid is freely movable and sufficient in amount to rise above the pelvis—two or more litres.

The method of determining fluctuation in circumscribed collections of fluid, as pancreatic or other cysts within the abdomen, circumscribed effusions, dropsy or empyema of the gall-bladder, etc., is somewhat different in technic and available only in patients whose belly walls are comparatively thin. The tips of the palpating fingers are lightly placed in contact with the surface at one border of the area under examination while the opposite border is sharply but lightly flicked with the nail—dorsal surface of the tip of the middle or ring finger suddenly disengaged from contact with the palmar surface of the thumb, as one flicks a crumb. By this method not only can fluctuation of limited extent be determined but also the limits of the area in which it is present defined.

Pulsation, Thrill and Fremitus.—Pulsation.—In thin persons the normal pulsation of the aorta may be felt upon deep palpation in the middle line about the level of the umbilicus. Abnormal pulsation of the abdominal aorta is of two kinds, the so-called dynamic pulsation seen in neurotic persons, not expansile and not associated with tumor or other signs of dilatation of the vessel, and the expansile pulsation of abdominal aneurism, in which a tumor that can be grasped between the hands and is the seat of distinct expansile pulsation may be present together with other signs of aneurism. The differential diagnosis between these two forms of pulsation should not be a matter of doubt. Pulsation is sometimes transmitted from the aorta to a tumor overlying it in such a manner as to simulate aneurism, especially as the pressure of the tumor may cause both bruit and thrill. The fact that the pulsation is not expansile and the palpation of the tumor in the knee-elbow posture, when the movement of the aorta is no longer communicated to it, serve to render the differential

diagnosis between such a tumor and aneurism a matter of comparative ease. Dynamic pulsation of the aorta is felt in the course of the vessel in the middle line and slightly to the left of it; that of aneurism is usually more extended transversely and may be felt some distance to the left, even reaching almost as far as the iliac crest, as I saw in a case verified by autopsy.

The liver pulsation due to tricuspid incompetency—hepatic venous pulse—may frequently be recognized upon palpation, especially bimanual palpation, when it is not visible upon inspection, and by the former method the difference between the expansive movement of a pulsating liver and the jogging due to the communicated movement of the heart may be appreciated.

Thrill.—This sign is sometimes met with in abdominal aneurism and tumors pressing upon the aorta. It has little diagnostic significance.

Fremitus is the sign of echinococcus cysts—hydatid fremitus or thrill. The tumor is soft, elastic, fluctuating, and in the majority of cases the seat of a peculiar vibration or fremitus, which may be felt by palpation with two or three fingers of the same hand or by placing three finger-tips widely separated upon the surface and lightly percussing the middle finger. Gall-stone fremitus is sometimes elicited upon palpation of the gall-bladder distended with a large number of calculi. It is a comparatively rare but very important sign.

Movements of Abdominal Organs or Tumors.—The movements of intra-abdominal organs and tumors constitute physical signs of great value in diagnosis. They are *respiratory, postural, and manipulative.*

Respiratory movements are communicated to the organs in close relation to the diaphragm, especially the liver, spleen, and to a less extent the kidneys. Tumors of the stomach are usually but little influenced by the movements of the diaphragm. Conditions which hinder the respiratory movements of the diaphragm, such as pleurisy, emphysema, massive enlargement of the liver or spleen, advanced pregnancy, meteorism and ascites, restrict or wholly arrest the respiratory movements of abdominal viscera. The anatomical relations of the pancreas and retroperitoneal glands are such that they are not influenced by the movements of respiration. Very large cysts of the pancreas may show slight movement on deep breathing.

Intra-abdominal new growths which are influenced by respiratory movements originate in the upper portion of the cavity; those which manifest no respiratory movement upon careful palpation commonly but not always develop from the pelvic organs or from structures directly connected with the spinal column behind the peritoneum—pancreas, retroperitoneal lymphatic glands, aneurism.

Postural Movements.—Free fluid in the cavity tends to gravitate to the most dependent space, while the air-containing intestines float upon the surface of the fluid. Small effusions may cause dulness in the umbilical region when the patient assumes the knee-elbow position. Floating viscera, kidneys, spleen, and in very rare instances the liver, are recognized upon palpation by their size, shape, and general relationships. A liver dislocated has little range of movement, but the kidney and spleen may be found in distant regions of the abdominal cavity, even at the brim of the pelvis.

The Technic of Palpation of the Kidney.—The recognition of a displaced kidney is not attended with difficulty. Palpation should be bimanual, one hand pressing upward from the lumbar region while the other is gently moved over the anterior surface of the abdomen, which should be as relaxed as possible. The tumor is oval, smooth, firm, and has the oblong shape of the kidney. It is sometimes possible to recognize the hilum and to feel the pulsating renal artery. The tumor is usually sensitive to firm pressure and freely movable. In the knee-elbow posture it advances towards the wall of the abdomen, while it sinks backward and may be pressed into its normal position when the patient assumes the dorsal decubitus. In the lateral and erect postures, it sinks to the lowest point of its range of movement. Except in the case of a much elongated mesonephron, it moves also with the movements of respiration. Wandering kidney is more common in women, in multiparæ, upon the right than the left side and is occasionally bilateral.

The Technic of Palpation of the Spleen.—The patient should be placed in a position midway between right lateral and dorsal, with his left hand upon his head. The thighs should be flexed in order to relax as far as possible the abdominal wall. The head should be slightly retracted and the patient directed to breathe deeply and slowly. The physician, standing at the patient's right, exerts with his left hand firm pressure upon the infra-axillary region downwards and forwards while, with his right hand, he presses the soft belly wall below the arch of the ribs upwards and inwards to determine whether or not the lower border of the spleen can be felt and in particular at the end of deep inspiration. Too much force must not be employed lest a greatly softened spleen, as in enteric fever, might be ruptured. The data yielded by percussion in the examination of the spleen are rendered uncertain by gastrectasis, meteorism, pleural effusion, and fecal accumulations in the colon and new growths in the splenic region. The results of palpation in moderate enlargement are much more satisfactory and reliable.

The diagnosis of massive enlargement of the spleen is usually a matter of ease and certainty. The contour of the tumor, upon which may be distinctly felt a sharply rounded inner border, often notched opposite the hilum, its firmness, its slight movement upon deep breathing, and the smoothness of the surface are of diagnostic importance.

Wandering spleen is not often difficult of recognition. The displaced organ is readily palpable below the left hypochondrium, less often in the umbilical or left iliac region, and very rarely at the brim of the pelvis, as a smooth oval tumor of the outline of the spleen, notched and freely movable upon change of posture and by manipulation. If the organ occupies a position to which the movements of the diaphragm do not extend, it does not move even upon the deepest respiration.

Movements upon Manipulation.—All abnormal organs and tumors that change their position in response to changes in posture are movable upon manipulation or palpation. The list comprises floating liver, spleen, and kidney; in the absence of adhesions, tumors of the pylorus and less frequently of other parts of the stomach, as the greater curvature, new growths in the intestines, excepting the ascending and descending colon;

fecal accumulations, gall-stones and enteroliths; mesenteric and omental tumors. The range of movement is limited in tumors of the gall-bladder and pancreatic cyst, in the upper regions of the abdomen; very limited in tumors of the ascending and descending colon laterally and enlargements of the uterus and ovaries in the lower segment. All malignant and some benign tumors tend to contract adhesions which interfere with movement. The following are immovable. small tumors of the pancreas, retroperitoneal growths, peri-appendiceal infiltration, adhesions and abscess, abdominal aneurism and abscesses.

Peristaltic and Fetal Movements. — The peristaltic movements may sometimes be felt, as they may be seen, in thin individuals in health and when in obstruction of the bowel they become excessive. In chronic, slowly developing stenosis of the gut the musculature of the intestines undergoes hypertrophy and the peristalsis becomes proportionately more powerful. Antiperistaltic or reverse waves may sometimes be felt. Palpable coarse intestinal movements with the formation of knots accompanied by borborygmi may be present in colic and in hysteria. The gastric and intestinal movements are very plainly felt and seen in cases of wide separation of the recti in women who have borne many children.

The movements of the fœtus may be often plainly felt upon palpation, and in advanced pregnancy the position of the fœtus may be recognized by this method of examination. All these movements may be rendered more active by manipulation and the sudden application of cold.

Outline.—By palpation we determine whether an intra-abdominal mass is round, oval, or irregular in outline; whether it is rough, nodular, or smooth; whether it resembles a viscus as the kidney or spleen in shape and has characteristic anatomical features, as the hilum or a pulsating artery. We ascertain its apparent point of origin, as in the epigastrium, the lateral regions of the abdomen, or the pelvis, and whether or not it has direct attachments or relations with another organ, such as may be made out between an enlarged gall-bladder and the liver, carcinoma of the pylorus and the stomach, or a large cyst in the left hypochondrium and the pancreas.

Density and Elasticity.—The signs relating to the consistency of an intra-abdominal mass can be ascertained by palpation alone. We thus determine whether it is fluctuating as in abscess or cyst; soft as in rapidly developing new growths and aneurism; moderately firm as in organs the seat of congestion and hypertrophy, or dense as in slowly developing carcinoma or interstitial overgrowths. In fecal accumulations the tumor is sometimes hard and firm, sometimes soft and doughy and can be indented by the finger.

Surface.—Palpation enables us to study the surfaces of organs and tumors. The smooth surface of an amyloid or fatty liver, the coarse granular surface of the liver in atrophic cirrhosis, the nodular liver with its rounded isolated eminences at the summit of which slight depressions may be felt—Farre's tubercles—in cancer, are examples of surface changes of diagnostic importance. The smooth surface of the distended gall-bladder stands in strong contrast with the irregular outline of carcinoma of the pylorus; the irregular multilocular echinococcus of the liver can hardly be differentiated from hepatic cancer, but is wholly unlike the smooth, elastic, and vibrating single

hydatid cyst. The smooth, elastic, and fluctuating cyst in hydronephrosis differs altogether from the firm, nodular and irregularly shaped mass in carcinoma of the kidney; and the smooth, ovoid, nearly centrally placed tumor of early pregnancy is wholly unlike the tumor formed by irregular, coarsely nodular subperitoneal uterine myomata.

MENSURATION.

Instruments of Precision—Graphic Methods.

The use of instruments of precision is of great importance in physical diagnosis. Such appliances vary from a simple graduated tape to the most intricate and delicate hæmodynamometer or polygraph. The writer holds the opinion that simplicity both of method and of instruments yields the most satisfactory results at the bedside, and that intricate and costly mechanical devices which require great technical skill and consume much time are better suited to scientific research than to every-day clinical work.

Measurement of the chest—thoracometry—may be conveniently made by a steel tape graduated upon one side in centimeters, on the other in inches; the diameters are taken by calipers made for the purpose.

The circumference and semicircumferences are taken at the level of the nipples or the fourth costosternal articulation in quiet breathing, in full held inspiration and on full expiration. Care must be taken that the tape is horizontal. The normal chest is nearly but not quite symmetrical, the right semicircumference being in the majority of individuals slightly larger than the left—an average difference of about half an inch. It is well to make a mark with a dermatographic pencil in the median line in front and over a vertebral spine at the same level and measure the semicircumference from point to point on each side for comparison. Two tapes attached to a little wooden saddle which fits over a vertebra are useful to determine the semicircumference on quiet breathing and the differences on forced respiration. The average circumference in men is 34.3 inches (87 cm.); in women 29.5 inches—(75 cm.). The difference in forced expiration and full held inspiration varies in normal individuals between 1.5 (4 cm.) and 5 inches (12.5 cm.).

The main diameters of the chest at the same level as taken by compass calipers with curved arms or slide calipers are: anteroposterior (the depth of the chest) average in repose in men 7.5 inches (19 cm.); in women 6.9 inches (17 cm.); bilateral or transverse (the breadth of the chest), average in men 9.9 inches (25 cm.).

Spirometry.—By this means we ascertain the volume of the tidal air. The instrument used is the spirometer. Various forms are in use, but the results are far from satisfactory. The instruments are cumbersome and require a certain amount of training to obtain constant results. The sex, age, weight and height must be taken into account. Thus for every inch above five feet, eight cubic inches are to be added to the normal standard, which for five feet is 17 cubic inches. The estimated average lung capacity for height in males between sixteen and forty years of age is, according to Otis,

CYRTOMETRIC TRACINGS.

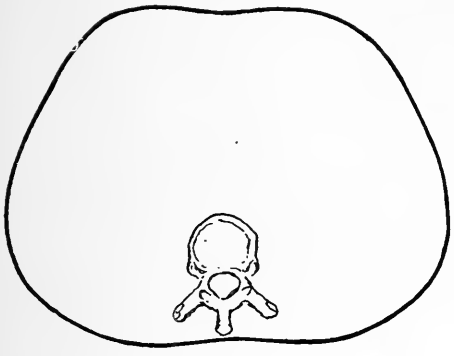


FIG. 48*a*.—Outline of normal chest.

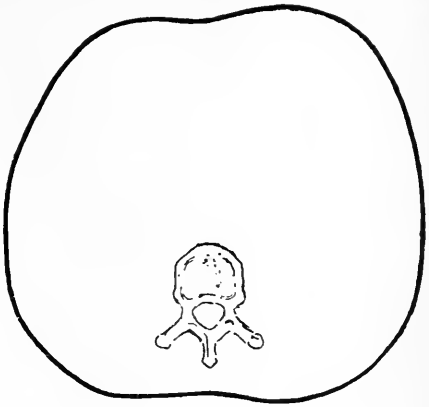


FIG. 48*b*.—Outline of emphysematous chest.

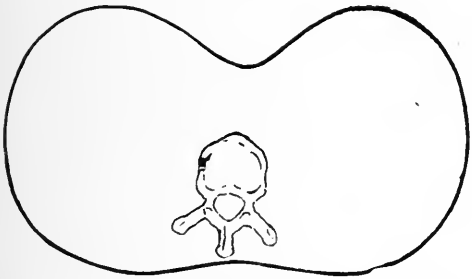


FIG. 49*a*.—Outline of chest showing "funnel shaped" deformity.

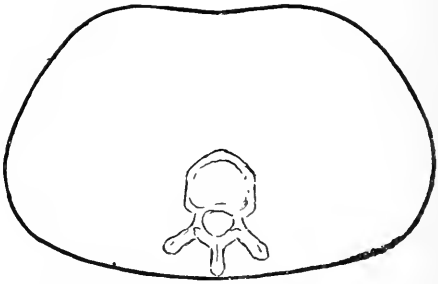


FIG. 49*b*.—Outline of phthisical chest.

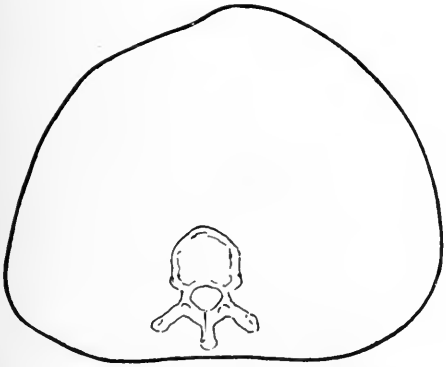


FIG. 50*a*.—Outline of the chest in spinal curvature.

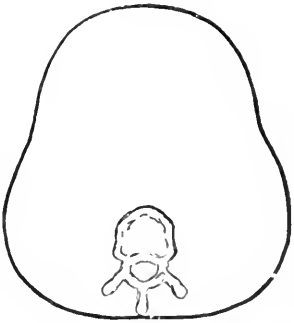


FIG. 50*b*.—Outline of the chest in rickets.

twenty-three cubic centimeters for every centimeter of height; in females at nineteen years of age, it is fifteen cubic centimeters for each centimeter of height.

Waldenburg's pneumatometer is an apparatus designed to measure the respiratory energy. Normally the power exerted in expiration is greater than in inspiration by from twenty to thirty millimeters of mercury. In emphysema and asthma the expiratory pressure is greatly diminished, while in certain forms of phthisis the inspiratory power is much lessened.

Crytometry.—The determination of the outline of a cross-section of the chest may be made with an instrument called a cyrtometer—measure of the curve. This procedure is of no great use in ordinary clinical work but very suggestive and important in teaching. Elaborate and costly instruments are not necessary for this purpose. The best device consists in a little metal saddle made to fit the spine, to each side of which is hinged a strip of leaden ribbon half an inch in width and thick enough to be easily bent so as to conform to the surface of the chest, yet retain its form when removed. The saddle is set upon the spine at the level selected, the leaden band is carefully adjusted to the surface on each side and made to meet at the median line in front. It is then released, opened at the hinges, removed from the chest and then laid upon a sheet of paper, the ends being brought together at the point of meeting in the median line. The outline is controlled by the fixation of the main diameters by means of the calipers. A soft pencil is then used to make the tracing on the inside of the cyrtometer. The various deformities of the chest described under inspection may be thus depicted.

Circumferential measurements of the abdomen at the level of the umbilicus and vertical measurements from the ensiform cartilage are useful, especially for purposes of comparison in ascites and enlargements from tumor or other conditions. They are best made with the ordinary graduated tape. Measurements from various fixed points upon the surface of the thorax or abdomen are necessary for purposes of record.

Methods of Recording the Circulatory Movements.[1]

The Sphygmograph.—The first instrument introduced into clinical medicine for the purpose of recording circulatory movements was the sphygmograph. By its use a single record, usually of the pulsation in the radial artery, is obtained. After many years of careful work with this instrument it has now been practically discarded. It has fallen into disrepute because it fails to yield the knowledge which is to-day considered essential for the proper understanding of many disturbances of the circulation. In fact, except for the sake of a permanent objective record, little is gained which can not be foretold by the well-trained palpating finger. The form of the curves obtained from the pulsation in the artery is so liable to alteration by the improper adjustment of the instrument, or by the anatomical conditions, that conclusions drawn from the record are uncertain. The sphygmograph may, therefore, be considered of but meagre clinical value and the instrument will not be further discussed.

[1] Contributed by G. Canby Robinson, M.A., as collaborator.

The Polygraph.—An important contribution to the study of circulatory disturbances was made when James Mackenzie introduced the polygraph into clinical medicine. This instrument allows two or more simultaneous graphic records of circulatory movements to be made. By recording the pulsations of the jugular vein and of the radial artery at the same time, the movements of the auricles and of the ventricles can be studied. The study of the move-

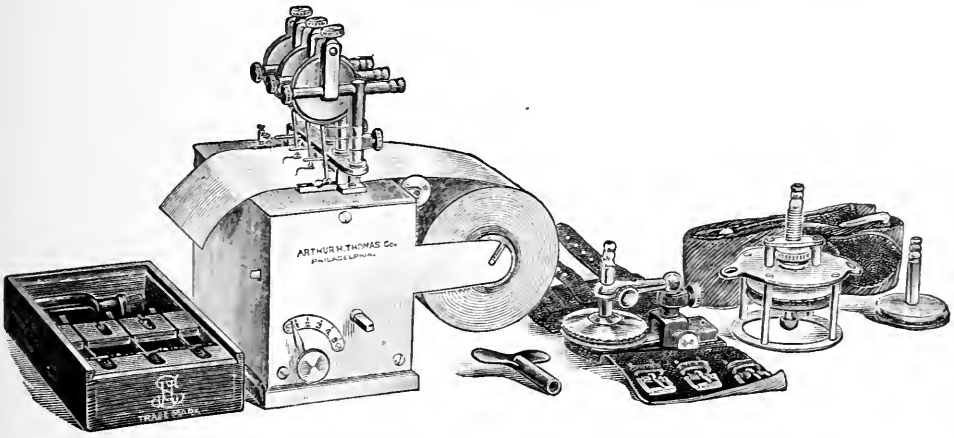


Fig. 51.—Jaquet portable polygraph.

ments of the two chambers of the heart has proved of great value in allowing the various types of cardiac irregularities to be differentiated.

Instruments have been devised which are readily portable and applicable at the bedside or in the office.

Two forms of instruments have come into general use for this purpose:

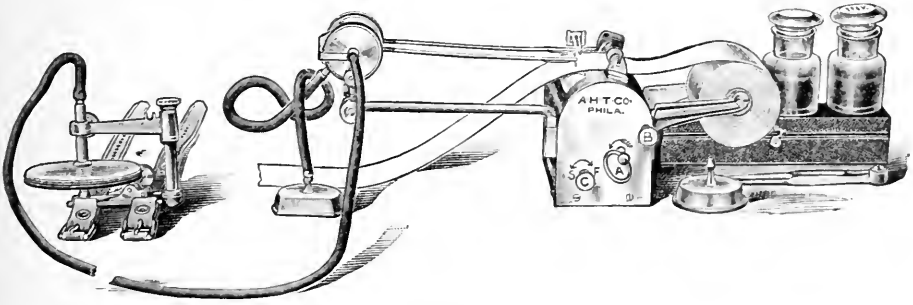


Fig. 52.—The Mackenzie ink polygraph.

the Jaquet polygraph, which records the pulsations of the radial artery and the jugular vein on smoked paper (Fig. 51), and the Mackenzie ink polygraph (Fig. 52). Each instrument is provided with a time-marker, marking off fifths of seconds so that intervals of distance on the records can be converted into intervals of time. The latter instrument is to be recommended for clinical purposes when used away from a laboratory, as smoked paper presents inconveniences. The Mackenzie instrument also allows prolonged records to be made while the Jaquet does not. An ordinary kymograph as

used in physiological laboratories, set upon a table on wheels, is serviceable for hospital use.

The study of the venous pulse record has laid the foundation for a clear understanding of the disturbances of the cardiac mechanism. The normal pulsations in the jugular vein yield a record consisting of three main waves (Fig. 53). These waves have been designated "A," "C," and " V " waves, because they are known as the auricular, the carotid and the ventricular waves. They bear a constant relation to one another and mark the definite activities in the cardiac cycle. The " A " wave is caused by an increased pressure in the jugular vein resulting from the contraction of the right auricle, which, while forcing the blood into the ventricle, also stops the outflow of venous blood and causes as well back pressure in the great veins. The " C " wave accompanies ventricular contraction, and at least in part is the result of the upward bulging of the tricuspid valves and the base of the auricles. The wave is often augmented by the pulsation in the

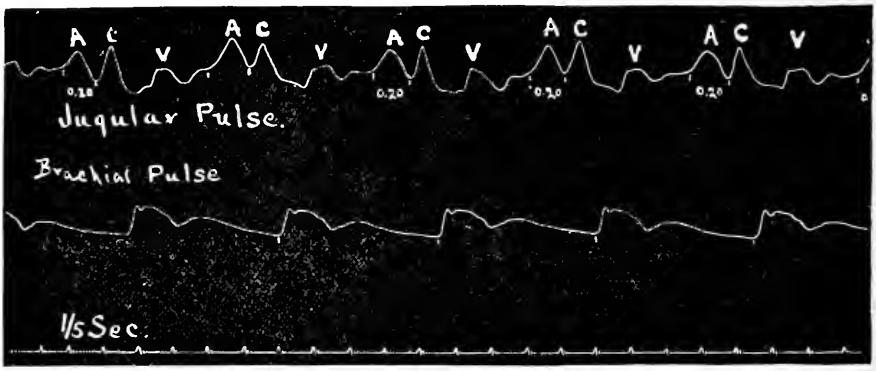


FIG. 53.—Normal record. Polygraphic tracing, the jugular pulse above and the brachial pulse below. Normal relation of A and C waves shown.

carotid artery which lies under the vein. The " V " wave occurs during the latter part of ventricular systole, the crest of the wave marking the completion of systole and the onset of diastole. The wave subsides when the tricuspid valves open and allow once more free passage of venous blood from the veins through the right auricle into the ventricle. Other waves have been described, but as they have but little bearing on clinical diagnosis they will not be discussed.

The interpretation of the venous pulse tracing is only possible with certainty when it is accomplished by a tracing from the radial or from some other artery. By comparison of the radial arterial tracing with the venous tracing, the " C " wave of the latter can be identified, as with each heartbeat it precedes the onset of the radial tracing by about one-tenth of a second, or half of one interval marked by the time-marker on the record. When the " C " wave of the venous tracing is identified, it will be found to follow the " A " wave in the normal record by about one-fifth of a second. The measurement is made from the onset or "foot-point" of each wave. It is the relation of the " A " and " C " waves which is of greatest value in

the interpretation of the cardiac action, as their normal relation is only disturbed when there is a lack of proper coördination of the auricles and ventricles of the heart. As nearly all forms of cardiac arrhythmia are accompanied by a lack of proper coördination of the auricles and ventricles, the question of this coördination is an important one. The polygraph will usually show whether there is a normal relation or not between the activities of the auricles and ventricles, and will indicate the various types of cardiac irregularities from one another. This differentiation is essential for the diagnosis, prognosis, and treatment of cardiac disorders accompanied by irregularities of the heart.

The analysis of polygraphic records obtained from cases in which various disturbances of the heart-beat are present is not difficult when the normal mechanism of the heart is kept in mind. It must be remembered that the impulse which sends the heart into contraction starts in the sinus node situated in the upper portion of the right auricle and spreads rapidly throughout the auricles, setting up a simultaneous contraction of both auricles. The impulse then passes from them through the auriculo-ventricular bundle (bundle of His) and reaches the ventricles after a definite interval of time. The impulse is then carried rapidly through the ventricular conducting system, and sets the two ventricles into simultaneous contraction. Any disturbance of rate of impulse conduction or of the normal sequence of the cardiac activity will be manifested by a disturbance in the coördination of the contractions of the auricles and ventricles, and will show itself in the record obtained by the polygraph.

The various disturbances of the heart-beat in which the use of the polygraph have been of especial value can be classified as follows:

Sinus arrhythmia,
Extrasystoles, or premature beats of the auricles,
Auricular flutter,
Auricular fibrillation,
Disturbances of conduction,
    Delayed conduction,
    Partial heart-block,
    Complete heart-block,
Extrasystoles, or premature beats of the ventricles.

Each of these types of disturbed cardiac mechanism can be distinguished by means of polygraphic tracings.

Sinus arrhythmia is caused by the irregular onset of the cardiac impulse, and shows itself in the venous pulse tracing by the occurrence at irregular time intervals of the group of waves which are seen with each normal cardiac contraction. There is in this condition no disturbance of the normal relations of auricles and ventricles, and therefore the "A" and "C" waves have throughout the normal relation to one another.

Extrasystoles or premature beats of the auricles are indicated in the venous tracing by the occasional occurrence of premature "A" waves, which are generally followed by "C" waves bearing the normal relation to them. This type of cardiac irregularity results from the premature contraction of the auricles, set up by an impulse generated somewhere in the

auricles themselves, before the rhythmical impulse from the sinus node reaches them. The impulse of this contraction is, as a rule, conducted to the ventricles in the usual manner, setting up also a ventricular contraction before the regular time for it.

Auricular Flutter.—The condition known as auricular flutter is one in which the auricles beat regularly at a very rapid rate, usually about three hundred times per minute, while ventricular contractions follow every second, third or fourth auricular contraction. The venous tracing which signifies that auricular flutter is present shows a series of rapidly recurring waves which may be rather poorly defined, and only every second, third, or fourth wave will be followed by the waves of ventricular contraction. These latter waves can be determined by comparing the venous pulse tracing to the tracing from the radial artery.

Auricular Fibrillation.—When the auricles go into a state of fibrillation, and so fail to contract as a whole, their activity is such that no definite waves are produced in the jugular tracing. Then the " C " and " V " are present without the normally preceding " A " wave, although a slight undulation may result from the auricular activity. The type of jugular trac-

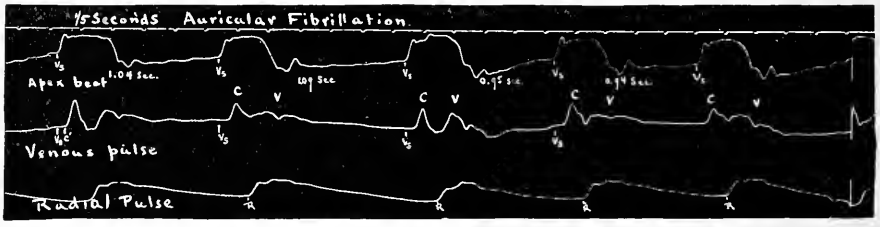


FIG. 54.—Auricular Fibrillation. Polygraphic tracing. Apex beat above, jugular pulse in the middle. Radial pulse below. No "A" wave is seen. The arrhythmia of the ventricles is indicated by the time intervals between each apex beat record.

ing is spoken of as the ventricular type of venous pulse. The well-defined waves that appear are unusually large, as a rule, and occur at irregular intervals. The irregularity of the heart is also indicated by the radial tracings and is characterized as being without rule or rhythm. It is known as the completely irregular pulse to which has been given the name *pulsus irregularis perpetuus*. The absence of the "A" waves and the complete irregularity of the occurrence of waves of ventricular activity serve to establish the diagnosis of the frequent and important form of disturbed cardiac action, auricular fibrillation (Fig. 54).

Heart-Block.—The disturbances of condition of the cardiac impulse from auricles to ventricles vary in degree from a mere delay to a complete cessation of conduction. If the passage of the impulse is delayed owing to a lesion in the conducting system between the auricles and ventricles, the normal time interval of one-fifth of a second between the onset of the "A" and "C" waves will be lengthened often to 0.25 or 0.30 of a second. If the damage to the conducting system is such that at times auricular impulses fail to reach the ventricles, "A" waves without the accompanying " C " waves will appear, and the condition of partial heart-block can be diagnosed from the record (Fig. 55). In this condition two "A" waves may appear

before each "C" wave, when the so-called two-to-one rhythm is present, or three "A" waves may be present before each "C" wave, indicating that only every third auricular beat succeeds in stimulating a ventricular contraction. If a mild grade of partial heart-block is present, there will be a gradual lengthening in the A-C intervals until finally an "A" wave fails to be followed by a "C" wave. This event is followed by a much shortened A-C interval, owing to the improvement in conduction resulting from the rest the conducting system has had. Then the gradual lengthening ending in another "A" without a "C" wave again takes place.

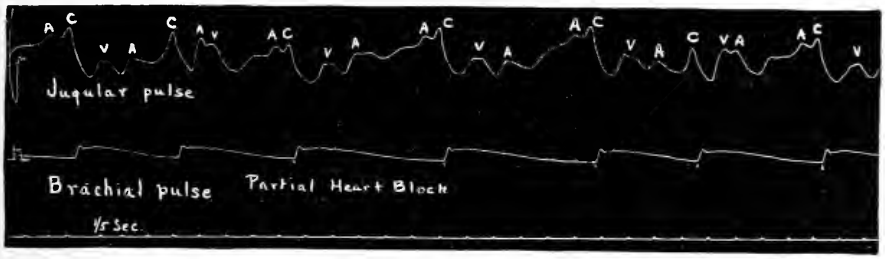


FIG. 55.—Partial heart-block. Polygraph tracing from the jugular vein and brachial artery showing two-to-one rhythm.

When there is complete heart-block, owing to a lack of physiological continuity between the auricles and ventricles, the "A" waves of the venous record will occur independently of the "C" waves, and at a much faster rate than the unusually slow rate of appearance of the "C" waves (Fig. 56). Such a record is characteristic of complete heart-block, and serves to explain the bradycardia which results from the slow rate of contraction which the ventricles maintain when beating independently of the auricles.

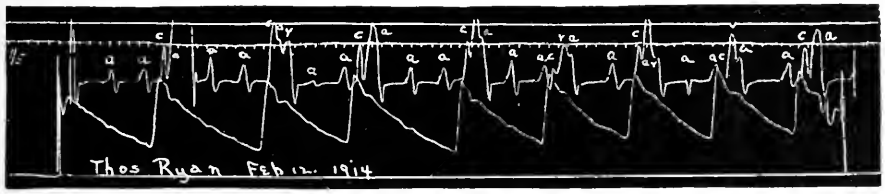


FIG. 56.—Complete heart-block. Polygraphic tracing, jugular vein above, radial vein below. Waves marked "a" represent auricular contractions.

Extrasystoles or premature beats of the ventricles occur when impulses arise in the ventricles independently of the normally descending cardiac impulse. This condition, resulting from an increase in the irritability of the ventricular musculature, may be thought of as "cardiac impatience," the ventricles refusing to wait for the normal impulse and going into contraction prematurely. These premature beats usually occur without disturbing the rhythmical auricular activity. The characteristic venous pulse yielded by such a disturbance of the heart shows a "C" wave occurring without a preceding "A" wave which may follow it and fall with the "V" wave or even after it. The "A" waves continue to occur at regular intervals, and those that fall near the premature "C"

waves are not followed, as a rule, by "C" waves, as the ventricles fail to respond on account of the contraction which they have just made. Following a premature ventricular beat or extrasystole, the ventricles remain in diastole until stimulated by the auricular contraction which follows that occurring just after the premature ventricular beat. As the auricular rhythm is undisturbed the ventricular rhythm is again reëstablished. This fact gives a certain regularity to the disturbance of rhythm caused by premature ventricular beats. The shortened diastole preceding the premature beat, and the lengthened diastole following it, are together equal to two regular cardiac cycles, as the length of both these intervals is determined by the regularly beating auricles. On this account the venous pulse tracing and the arterial tracing will show time intervals which are quite characteristic of ventricular premature beats or extrasystoles, and which are very helpful in the diagnosis of this common disturbance of the heart-beat. Premature ventricular beats may occur occasionally or very frequently, and at times they follow every normal ventricular beat that occurs, giving rise to the bigeminal pulse, every alternate ventricular contraction being replaced

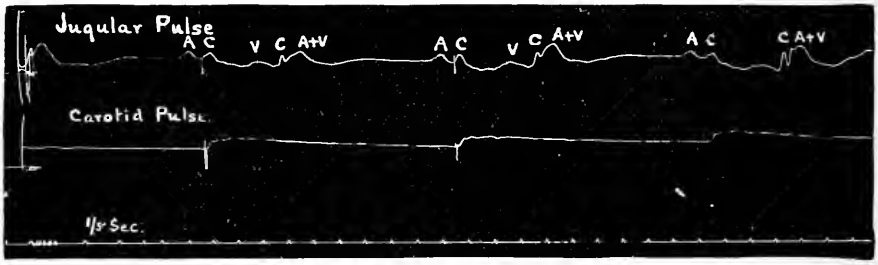


Fig. 57.—Premature ventricular beats or extrasystoles. Polygraphic tracing showing a premature beat after each normal beat. The premature beats produce practically no waves in the carotid pulse tracing.

by a premature beat. This condition is readily recognized by the study of the polygraphic record (Fig. 57).

Graphic records of circulatory activity are useful in recording the very rapid cardiac rate which occurs in paroxysm tachycardia, and which may be difficult to count with the palpating finger. Here the radial tracing alone suffices. They are also useful in recording pulsus alternans, in which weak and strong arterial pulsations alternate. This condition, which is indicative of a grave lesion of the myocardium, may give rise to alternating large and small waves in the radial record, and the venous pulse record serves to distinguish it from the bigeminal pulse caused by ventricular extrasystoles, which may be readily confused with pulsus alternans. This distinction is of importance on account of the great difference in prognostic significance of the two conditions.

The use of the polygraph requires considerable practice, and in the hands of the unskilled or of the impatient physician will be disappointing. An assistant is often necessary for its proper use, especially for a beginner: but persistence and practice will finally yield results which will warrant all the time and energy which may be necessary to perfect the technique.

The Electrocardiograph.—The introduction of the electrocardiograph for the purpose of studying the cardiac movements of man has greatly ex-

tended our conceptions of the normal cardiac functions and has shed a clear light on the various disturbances of the heart action. The credit for this achievement belongs to Einthoven of Leyden, who described this new instrument in 1903. It was not until about 1909, however, that several workers began to use the instrument and to apply it as a diagnostic aid in clinical medicine. At this time the first instrument was brought to the United States by Walter B. James and operated by H. B. Williams and himself. During the brief period of less than ten years it has proved of great value both for purposes of physiological research and for clinical study. The electrocardio-

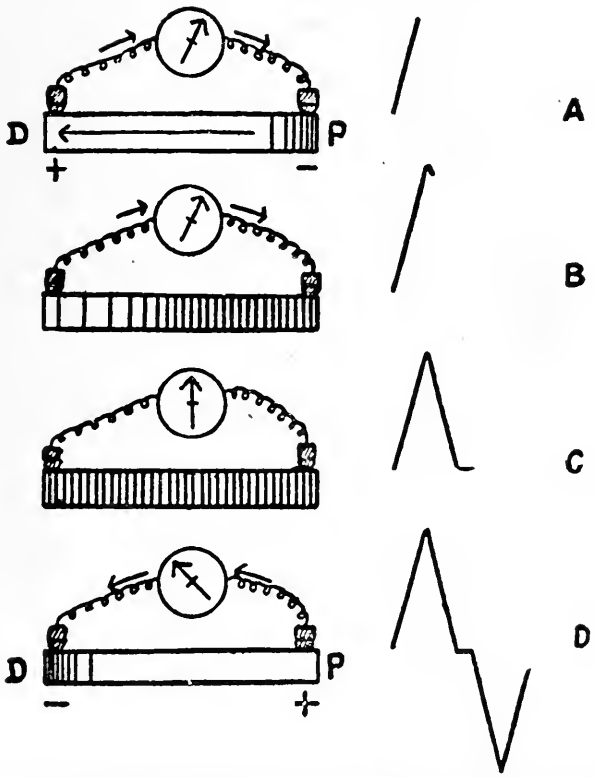


FIG. 58.—A diagram illustrating the development and subsidence of activity (and negative) in a single muscle strip, responding to a stimulus applied at *P*. The corresponding and successive phases of the galvanometric curve are shown in the four lines, *A*, *B*, *C*, and *D*.—(After Lewis).

graph has the disadvantages of being stationary, expensive, and requiring considerable training for its proper operation. It is, therefore, largely of use in hospitals. An understanding of the records obtained by this instrument is necessary, however, to read intelligently much of the recent literature dealing with cardiac disease. In the following discussion technicalities will be included only so far as necessary for an understanding of the records. For a complete explanation of the electrocardiographic method special books and papers on the subject must be consulted. (Lewis, Clinical Electrocardiography.)

The string galvanometer, that part of the electrocardiograph to which

all others are accessory, is an instrument for measuring and recording electrical currents, and its chief merits are its capability of indicating minute electric currents and of recording a rapidly changing series of currents. When a current passes through the very fine filament of the instrument, this filament is deflected to one side or the other, depending upon whether the current passes up or down the filament. In making an electrocardiogram the patient is put in circuit with this very delicate filament, which is moved back and forth by the series of currents which are generated by the contractions of the cardiac muscle. It is a well-known phenomenon of muscular contraction that the point in any muscle at which a contraction starts has a negative electrical potential when compared with the uncontracted part of the muscle. If a conductor, such as a wire, is attached to the two ends of the muscle, a current will pass from the part which is at rest to the part which has gone into contraction, and will continue to pass until the whole muscle has gone into contraction, when the electrical potential becomes again equalized, just as it was when the muscle was at rest. When the part of the muscle which first went into contraction now relaxes, a current will pass through the conductor in the opposite direction, because again one end, now the opposite end, of the muscle is electrically negative to the relaxed, non-contracting end. When relaxation is complete no more current passes. These facts are diagrammatically shown by the accompanying figure taken from Lewis (Fig. 58).

A point to be borne in mind is that the electric currents which are generated by the contractions of the heart muscle become evident only when a conducting system is led from one point on the body-surface to another and the recording apparatus is placed in the circuit. The conducting system is so attached to the body that one contact leads off from the base of the heart while the other leads off from the apex. As, of course, the contacts can not be made directly on the heart in man, the body tissues themselves are used as the conductor between the heart and the actual point of contact. The two arms and the left leg are used as the points of contact, and wires are attached to these parts by means of easily applied conductors. In making the records, of course, but two contacts are used at a time, but by first using the two arms, then the right arm and left leg, and lastly, the left arm and leg, the three usual first, second, and third leads are obtained. Currents pass back and forth from these points of contact during each cardiac contraction, and so the string of the galvanometer is moved.

When the heart is beating normally a current is generated, first by the contraction of the auricles, which causes a characteristic deflection of the string, then, after a short pause, during which time the impulse of contraction is passing down from the auricles, the ventricles go into contraction, and a series of movements is set up. The complexity of these movements is due to the complexity of the muscular arrangement of the ventricles and the complicated muscular contractions that go to make up ventricular systole. The movements of the string are recorded by throwing its shadow upon a moving photographic film or paper, the shadow of a time-marker being thrown upon the recording surface at the same time. The movements of the string are in this way converted into a record which consists of a

series of waves. These waves are usually five in number, three positive and two negative, designated arbitrarily P, Q, R, S, and T. The P wave represents the auricular contraction and can be readily distinguished from the Q, R, S, and T group that represents the ventricular contraction (Fig. 59).

The length of the circuit from one side of the patient's body to the string galvanometer and back to the other side of the body has practically no influence on the type of record obtained. For this reason it is not neces-

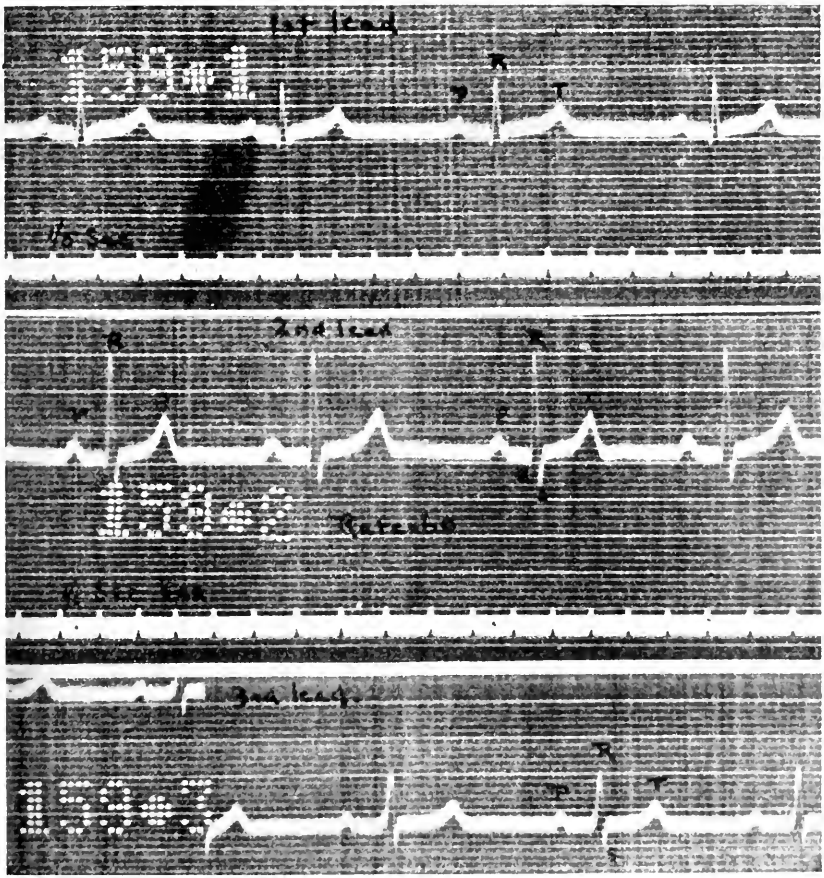


FIG. 59.—Electrocardiogram of a normally beating heart.

sary to have the patient near the galvanometer, provided wires can be led between the patient and the instrument.

The fact that in the electrocardiogram the record yielded by the auricular activity can be readily distinguished from the record of ventricular activity is of great importance, and allows the various disturbances of the heart-beat to be identified with certainty. The form of the record produced by both auricular and ventricular activity is altered whenever the passage of the cardiac impulse follows an abnormal course, so that striking changes in the form of the curves occur whenever a cardiac impulse arises in an

abnormal point and so courses over the heart along abnormal paths or in an abnormal direction. Thus the occurrence of a so-called ectopic impulse is recognized, whether it arises in the auricles or in the ventricles. Changes in the form of the curve also take place whenever the muscle mass on the two sides of the heart, especially in the ventricles, is disturbed by hypertrophy.

The electrocardiographic records obtained from the commoner forms of deranged cardiac mechanism will be discussed. They are best classified by the various regions of the heart affected, and include the disturbances of the (a) sinus region, (b) the auricles, (c) the auriculo-ventricular conducting system, and (d) the ventricles.

The disturbances in these various parts of the heart may be taken up just as they have been when the polygraph was under consideration.

(a) The activity of the sinus region, where the cardiac impulse arises, is not indicated in the human electrocardiogram, so that any disturbance of impulse formation in this region is only indicated by the irregularity at which the normal auricular and ventricular waves appear. When the

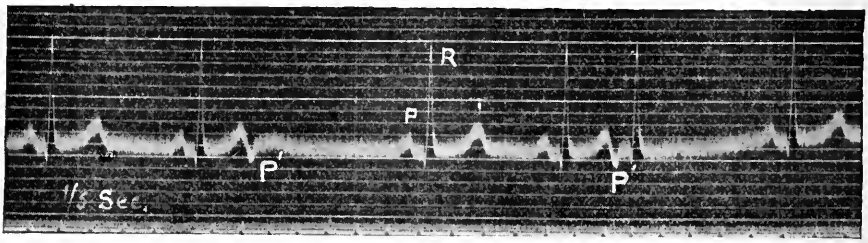


Fig. 60.—Premature ectopic auricular beats or auricular extrasystoles. Electrocardiogram shows two downwardly directed auricular waves, occurring prematurely. They indicate that the impulse has arisen near the base of the auricles. Only one of the two ectopic beats stimulates a ventricular contraction.

diastolic portions of the electrocardiogram vary from beat to beat, sinus arrhythmia is present, a condition which is in itself without pathological significance.

(b) The contraction of the auricles yields a small, rounded, characteristic, upward wave when the auricles are stimulated by an impulse from the sinus region. The form of the wave is quite changed if the impulse arises elsewhere. If it should arise near the lower part of the auricles instead of in the sinus region, it would travel upward instead of downward, and the auricular musculature would pass into contraction in a direction opposite to the normal. This ectopic contraction would yield a downwardly directed wave instead of an upwardly directed wave. This impulse reaching the sinus region would disturb the rhythm of impulse formation normally taking place there, and an irregularity of the heart would result (Fig. 60).

Impulses may arise from an ectopic focus at a very rapid rate, often at about two hundred times a minute, thus giving rise to tachycardia of auricular origin, such as occurs in paroxysmal tachycardia. This condition yields a characteristic record, recognized particularly by the high cardiac rate.

Another type of disturbed auricular activity is that known as auricular flutter, in which the auricles contract with a still higher rate. The auricular

rate may be over three hundred per minute, and when such a rate is attained, it is usually accompanied by a varying degree of partial heart-block, only every second or third auricular contraction sending an impulse to the ventricles (Fig. 61).

Auricular fibriliation, the most frequent type of disturbed cardiac mechanism in patients with broken cardiac compensation, is clearly demonstrated by the electrocardiogram. In this condition the auricles no longer contract as a whole, but the various muscle bundles composing the auricular walls contract and relax independently of one another, giving rise to a

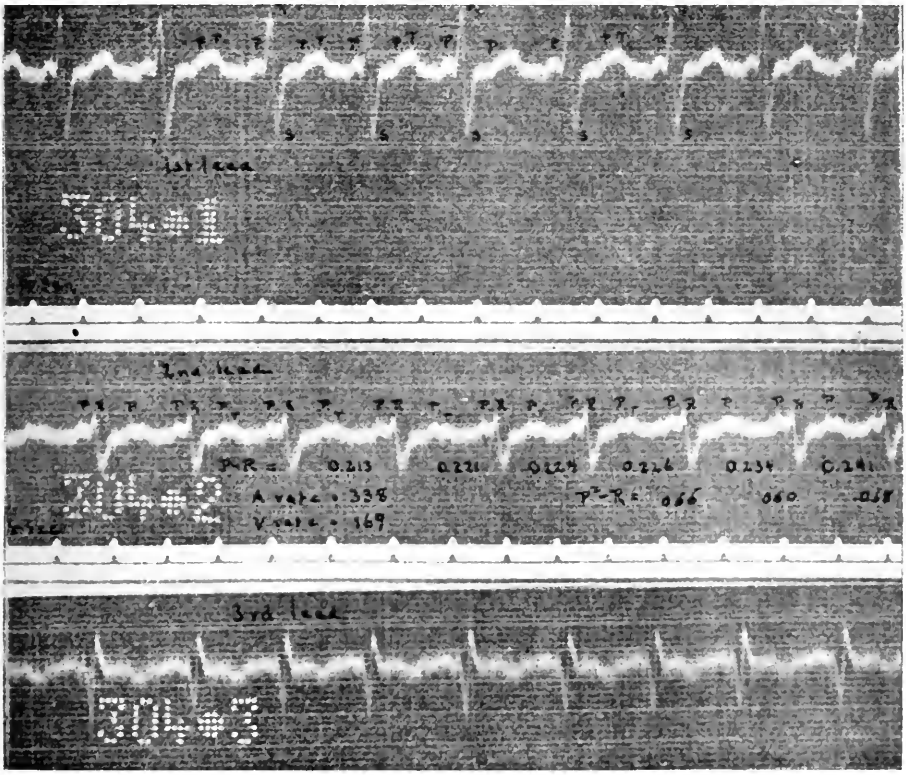


Fig. 61.—Auricular flutter. Electrocardiogram in which the auricular waves occur at a rate of 338 and the ventricular waves at a rate of 169 per minute.

chaotic, incessant activity in the auricles. This causes the ventricles to contract at irregular intervals, and is responsible for the absolutely irregular pulse. Electrocardiograms from patients with auricular fibrillation fail to show the regularly recurring series of "P" waves of auricular contraction which normally precede each group of waves of ventricular origin. Instead there is a series of small irregular waves which are seen in all parts of the curve representing ventricular diastole. The occurrence of these small irregular waves, together with the irregular occurrence of the ventricular group of waves, constitutes the typical electrocardiogram of auricular fibrillation (Fig. 62). The electrocardiogram is the clearest and most definite means of recognizing this condition.

(c) Disturbances of conduction of the cardiac impulse from auricles to ventricles are shown by changes in the relation of the auricular and the ventricular waves. If the disturbance is such that the cardiac impulse is delayed in its passage from the auricles to the ventricles, the interval between the beginning of the "P" wave (auricular) and the beginning of the "Q" or "R" wave (ventricular) is lengthened. Normally the so-called P-R time is 0.17 second or less, and whenever it exceeds this time

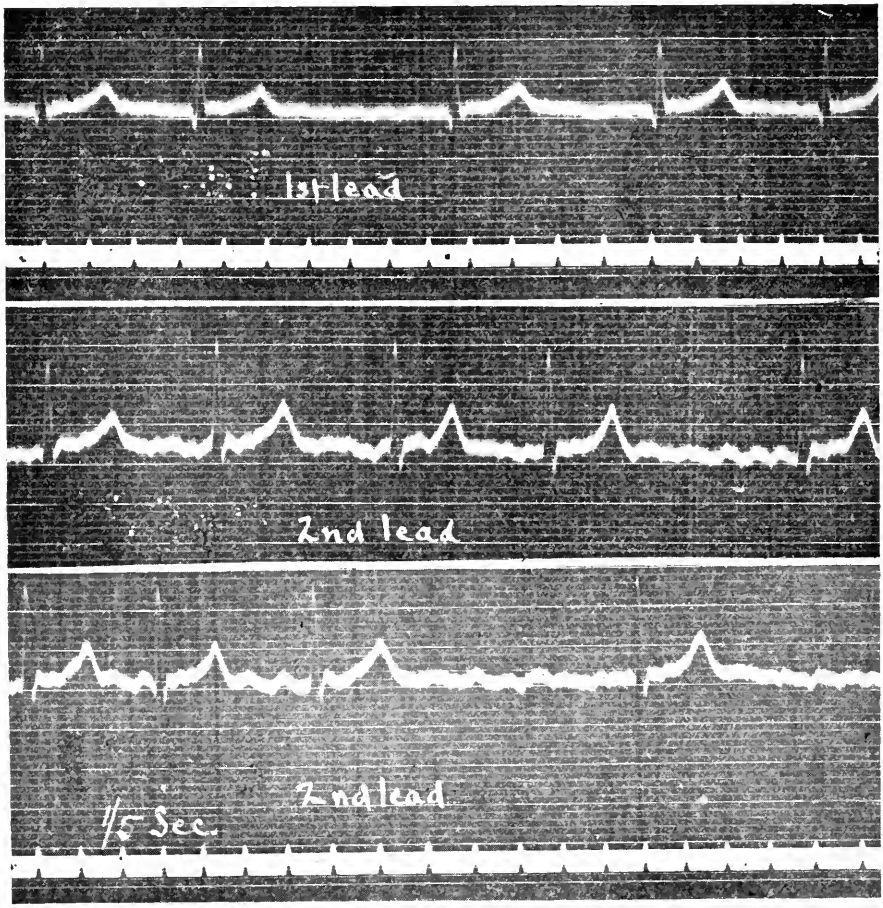


FIG. 62.—Auricular fibrillation. Electrocardiogram showing the absence of "P" waves, the irregularity of the ventricular waves, and the small irregular waves yielded by the fibrillating auricles during ventricular diastole.

there is a delay in conduction. By this means slight impairment of the auriculo-ventricular conducting system (the bundle of His) is revealed (Fig. 63).

When the ability to conduct the cardiac impulse from auricles to ventricles is further impaired, various grades of heart-block occur. This may result in an occasional auricular beat that is not followed by a ventricular contraction, or the defect in conduction may be so great that only every second or third auricular contraction sets up a ventricular beat.

These conditions are recognized at a glance when an electrocardiogram is obtained, the "P" waves of auricular origin being readily distinguished from the tall "R" waves of ventricular origin (Fig. 64). If there is a complete physiological separation of the two parts of the heart resulting in complete heart-block, the record shows the waves of auricular and ventricular origin occurring quite independently of one another and at an entirely different rate. Not infrequently the ventricular complexes have an abnormal form, probably indicating that the passage of the cardiac impulse from its origin in the basal portion of the ventricles is being hindered in its passage

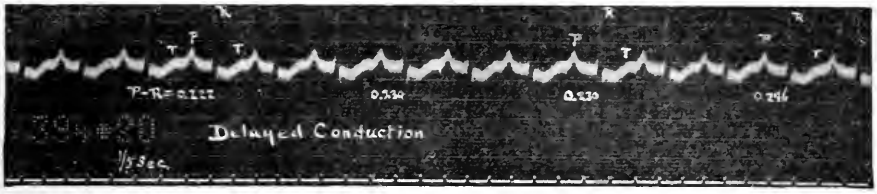


Fig. 63.—Delayed conduction. Electrocardiogram showing prolongation of P–R time marked in seconds.

through the ventricles, as the result of the same lesion which prevents the passage of the cardiac impulse from auricles to ventricles (Fig. 65).

(d) A very common form of cardiac irregularity is the result of the spontaneous generation of impulses in the ventricles, which set up premature ventricular contractions. When this occurs the impulse travels through the heart in an abnormal direction and along abnormal paths. This type of cardiac contraction yields, therefore, an abnormal form of electrocardiogram and the form will vary according to point of origin of the impulse. If it arises in the right ventricle it has a form which distinguishes it from an impulse arising in the left ventricle. As the cardiac contractions occur before

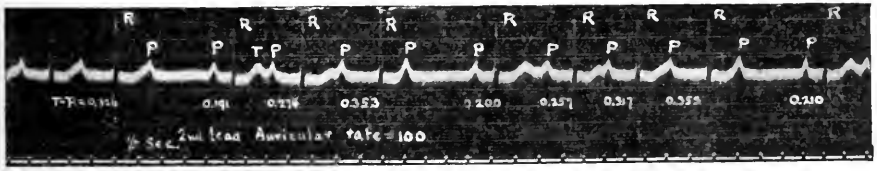


Fig. 64.—Partial heart-block. Electrocardiogram showing "P" waves unaccompanied by "R" waves and progressive lengthening of the P–R intervals until the block occurs.

the regular time, they are spoken of as premature beats, and as the impulses arise in an abnormal point they are known also as ectopic. The term ectopic premature contractions is more descriptive than the older and better-known term extrasystoles. The electrocardiogram demonstrates not only the presence of premature ectopic ventricular beats but also shows the time relations which exist between this type of beats and the normal contractions. The auricular rhythm is not disturbed, but the auricular contraction, which usually occurs almost synchronously with the premature ectopic ventricular beat, fails to produce a ventricular response, as the refractory phase of the ventricles has not passed off when the impulse from the auricles reaches it. For this reason there is a pause in the ventricular rhythm, and no contrac-

tion takes place until the next auricular contraction sends down its impulse to the ventricles. The pause and the unusually short period of diastole which precedes the premature beat are, therefore, of the same duration as two normal cardiac cycles. This is a point well shown by electrocardiograms

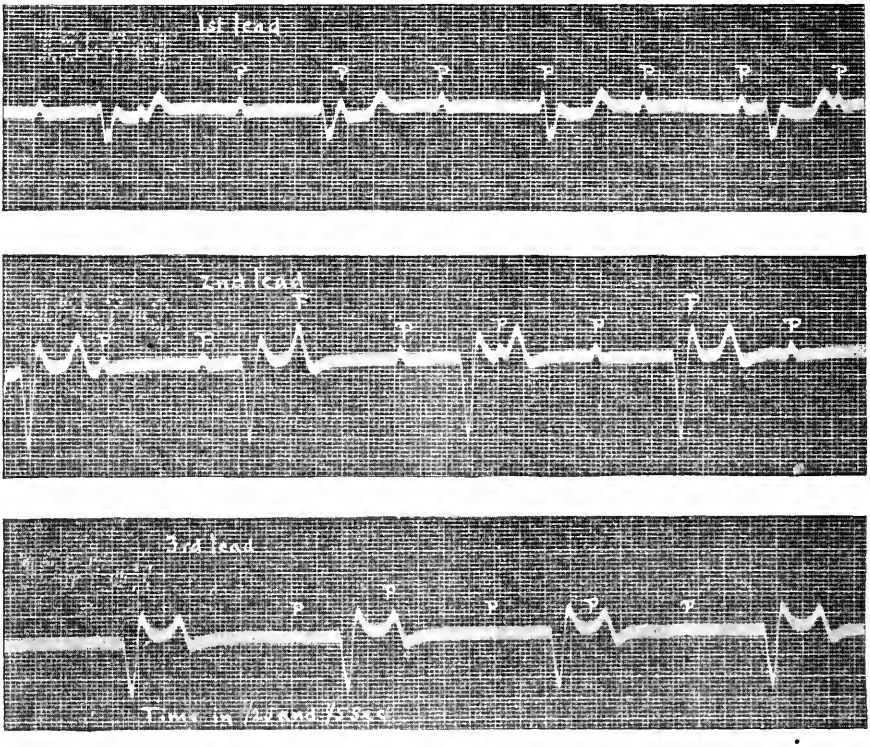


FIG. 65.—Complete heart-block. Electrocardiogram showing independent occurrence of auricular and ventricular waves. The ventricular portion of the record has a distinctly abnormal form suggesting disturbance of the passage of the cardiac impulse through the ventricles. From a child with diphtheria.

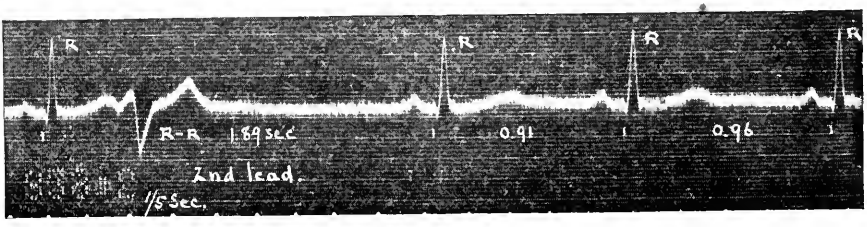


FIG. 66.—Premature ectopic ventricular beat. Electrocardiogram showing the abnormal type of ventricular record and the characteristic time relations.

and is useful in making a diagnosis of premature beats of ventricular origin whether this is done by the auscultating ear, the palpating finger, or by the study of electrocardiograms (Fig. 66).

The variations in the form of the ventricular portion of the electrocardiogram are significant, and may give important information regarding the physiological state of the ventricular musculature. This is especially

true when the records of the three usual leads are compared, as the relative height and direction of the main wave of the ventricular complexes in the three leads are altered by a relative increase of the muscle mass in the right ventricle as compared to that of the left. When the left ventricular musculature predominates, a change in the opposite direction in the electrocardiogram takes place. When the main waves, "R" waves, are compared in height in a normal record (Fig. 59) it is seen that they are upwardly directed

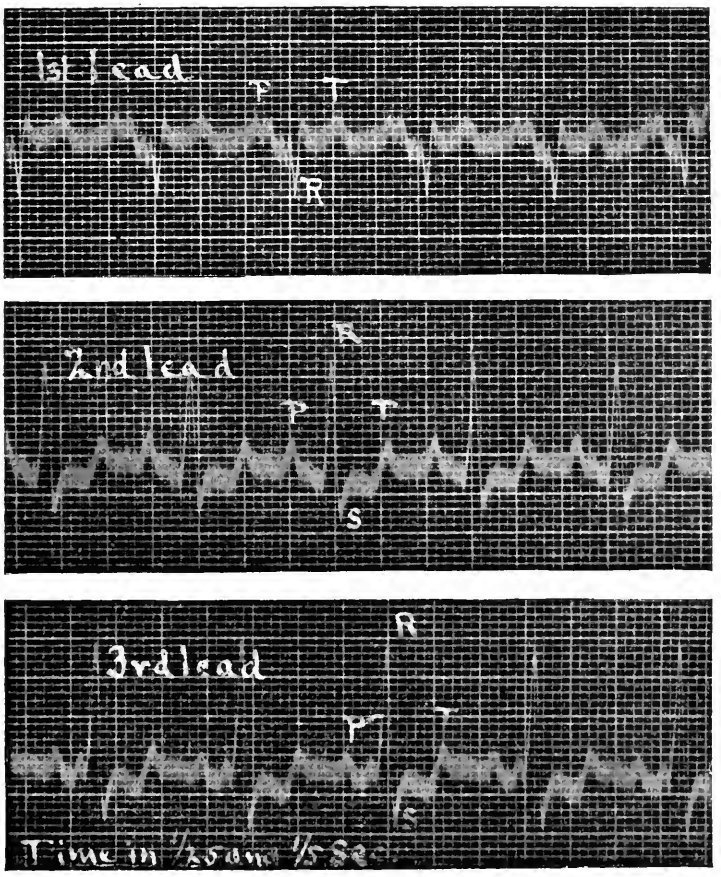


FIG. 67.—Hypertrophy of the right ventricle. Electrocardiogram showing the main wave of the ventricular record downwardly directed in the first lead and highest in the third lead. From a case of mitral stenosis.

in all leads, that the second lead is as tall as the first and third added together, and that the first is slightly taller than the third lead.

When there is hypertrophy of the right ventricle the first lead usually shows a downwardly directed main wave, while the third lead shows a main wave which is taller than that of the second lead. This relation of the three waves occurs practically exclusively in cases with mitral stenosis or congenital heart disease, and such a finding is frequently of distinct diagnostic value (Fig. 67).

With hypertrophy of the left ventricle the main wave of the first lead is tallest, while the main wave of the second and third leads may be downwardly directed that of the third lead always being deeper than that of the second lead. The electrocardiograms from patients with arterial hypertension or aortic insufficiency usually show such a relationship between the main ventricular waves of the three leads, and the degree of preponderance

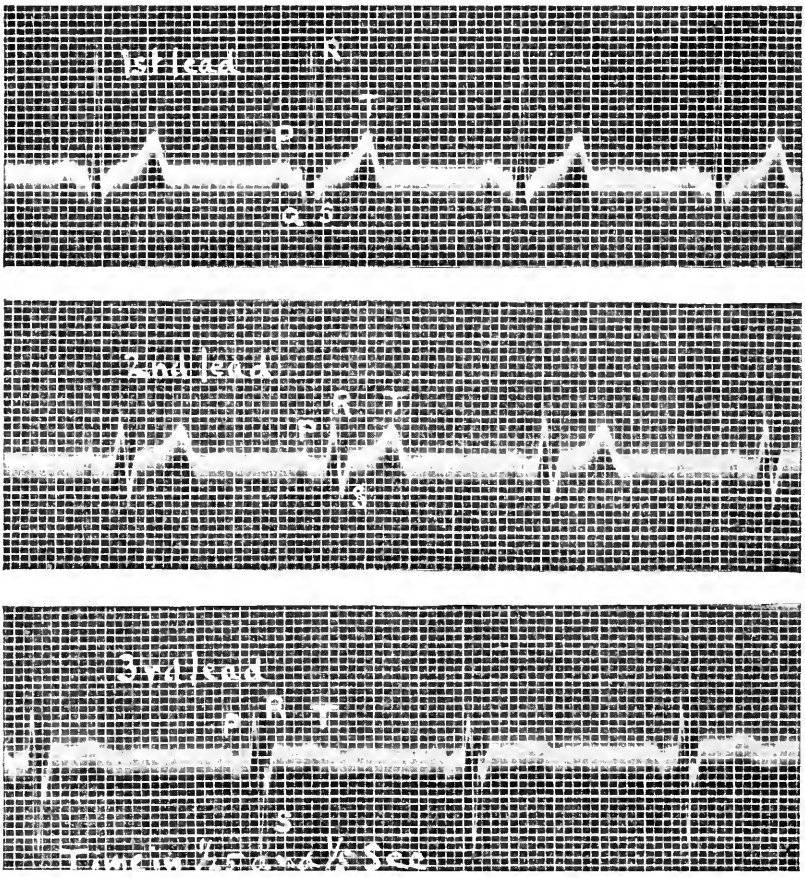


FIG. 68.—Hypertrophy of the left ventricle. Electrocardiogram showing the main wave of the ventricular record highest in the first lead and downwardly directed in the third lead. From a case of arterial sclerosis with hypertension.

of the left over the right ventricular muscle mass can be judged from the forms of the electrocardiograms (Fig. 68).

The interpretation of electrocardiograms of atypical forms presents a problem which is as yet not entirely solved, but to the experienced eye many abnormal forms of records are significant. Cases of damaged myocardium often yield records in which the final ventricular wave, the "T" wave, is downwardly directed in the first and second leads. These cases may also yield very small complexes, indicating that the electrical potential generated by the heart-beat is abnormally low. The atypical forms of electrocardio-

grams may be the result of delayed or blocked cardiac impulses while passing through parts of the ventricles. There may be a block in one or the other of the branches of the conducting system along which the impulse is conveyed to the ventricles, and when this occurs, a characteristic form of electrocardiogram results, varying with the position of the block. Disturbances of conduction in the less specialized parts of the ventricles may

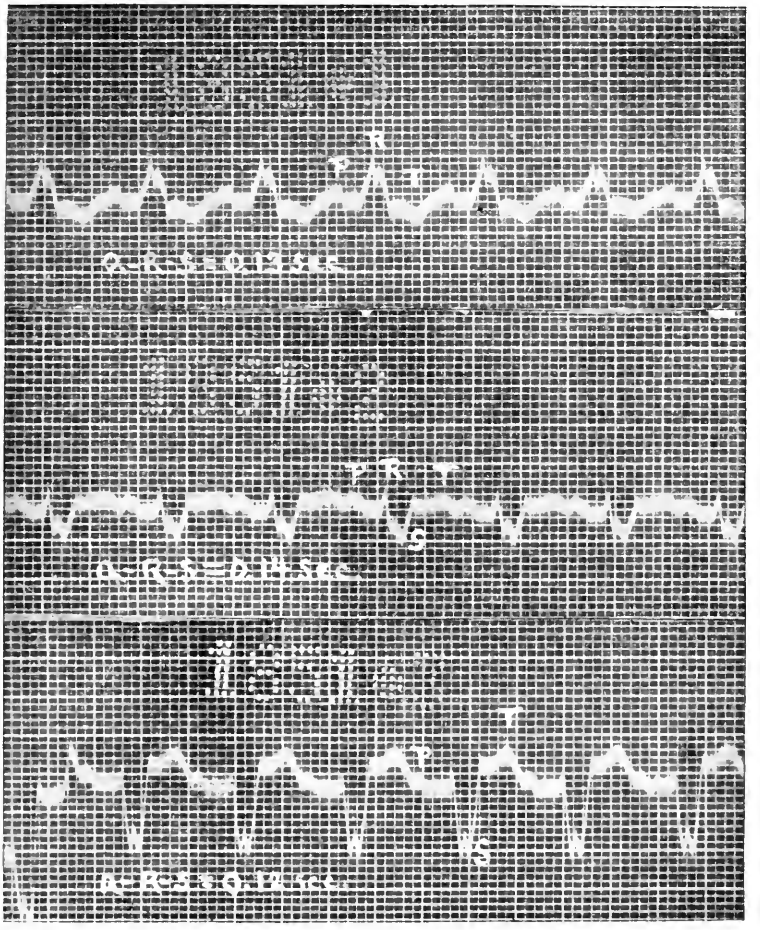


FIG. 69.—Electrocardiogram of abnormal form from a case of chronic myocarditis. Widening and notching of the Q-R-S group of waves in each lead, indicating that the cardiac impulse travels through the ventricles along abnormal paths or at an abnormal rate.

also be suspected from the form of the records (Fig. 69). The value of the electrocardiogram as indicative of the functional efficiency of the heart is not as yet definitely established, but the growing knowledge of the significance in form of the curves in the three usual leads is bringing a closer correlation between the functional activity of the heart and the form of the electrocardiogram.

Instruments for Measuring the Arterial Blood-Pressure.—Blood-pressure is the force exerted by the blood against the blood-vessel wall (lateral

pressure) or the force upon the blood current lying in front of it (end pressure). It depends upon four separate factors: (a) the energy of the heart; (b) the peripheral resistance; (c) the elasticity of the blood-vessel walls; (d) the volume of the circulating blood. One or all of these factors may vary under normal conditions. They may be greatly altered in pathological states.

The Systolic Pressure (maximal pressure) is the highest point reached in the artery during the ventricular systole. The Diastolic Pressure (minimal pressure) is the lowest point reached within the artery during the diastole of the ventricle. It is the peripheral resistance which the heart has overcome and which is maintained in the peripheral circulation during diastole. The Pulse Pressure is determined by subtracting the minimal from the maximal pressure. The average pressure during a certain period is called the mean pressure.

The Sphygmomanometer is an instrument used to determine the blood-pressure. The essential parts comprise a compressing armlet having a breadth of not less than 8 cm., non-distensile connecting tubes, an inflating apparatus and the manometer. Two types of the last are in general use, the mercury manometer and the aneroid manometer. The latter is more conveniently portable but requires occasional adjustment on comparison with an instrument of the former type. The newer instruments permit the determination of both the systolic and diastolic pressure. The mechanical principle is the indication by the manometer of the pressure necessary to cause obliteration of the arterial pulse. Clinically we measure the arterial pressure in the brachial artery, and the venous pressure in the veins of the hand or at the elbow. It is well to adopt some method to determine all pressures, thus eliminating error and loss of time. Preferably, the patient should be recumbent. The right or left arm may be used. The muscles should be relaxed since contractions are manifest on the manometer. The first estimation may be found 10 to 20 millimeters higher than subsequent readings, a variation probably due to excitement or fear of pain.

Repeated or control observations should be made. In cases of threatened circulatory failure it is, at times, practically impossible to get a clear-cut high or low pressure. The systolic pressures will vary from 5 to 15 millimeters even though repeated estimations are made. These cases may, at times, show a condition in which an occasional beat comes through at a higher level than that at which all beats can be detected. The respiratory action is often the causative factor. This should be noted thus—high pressure, occasional beat at 170; all other beats at 155.

A description of the various instruments supplied by the dealers is not deemed necessary since a complete explanation accompanies each apparatus.

The Palpatory Method.—The patient is placed in the recumbent or sitting posture with the muscles of the arm completely relaxed, the elastic cuff is closely applied to the bare arm above the elbow and distended by the means of inflating apparatus until the pulse can no longer be detected in the radial artery by the finger continuously applied; the pressure is now gradually reduced until the pulse reappears. The point indicated on the manometer is the systolic pressure. The diastolic pressure represents the greatest oscillation recorded by the manometer, either mercurial or aneroid, as the pressure upon the artery is carefully lowered. This procedure is probably

the best way of determining the maximal pressure, while the auscultatory method is surely the preferable one to ascertain the minimum pressure.

The Auscultatory Method.—The cuff is applied over the brachial artery, as already described under the palpatory method, and pressure in it raised until the radial pulse disappears. The bell of a binaural stethoscope is placed over the artery below the cuff without pressure. The air is gradually released and usually at the moment the pulse is felt at the wrist one will hear a slight tone which corresponds to the maximal pressure. As the pressure is further reduced the tones accompanying the pulse-beats grow louder and are sometimes accompanied by blowing murmurs. A point of maximal intensity is finally reached, at which junction the sounds suddenly become feebler and soon entirely disappear. This junction corresponds to the minimal blood-pressure.

Goodman and Howell have called attention to the remarkable cycle of auditory phenomena which correspond to the degrees of pressure as shown by the manometer and they have divided them into five distinct phases. When the pressure is normal—systolic 130 mm. and diastolic 85 mm.—these phases are well defined and bear a definite relation to the differences between the extremes of pressure. They are as follows:

1. A loud, clear, snapping sound ascribed to sudden distention of the artery by the inrushing blood. This sound is not unlike that of the first sound of the heart.

2. A series of hissing murmurs are heard accompanying the first phase sound, due to the formation of "fluid veins" as the blood flows through the constriction into the wider vessel beyond.

3. These murmurs disappear suddenly and a tone, usually much louder than that of first phase, is heard. It is the sign of reëstablished blood flow in the artery corresponding to the disappearance of the "fluid vein" due to the lessening of the pressure constriction upon the artery.

4. At the end of the third phase the sound suddenly becomes muffled in character. This point corresponds to the minimal pressure in the artery.

5. The fourth phase is very short and at the end of it all sounds cease.

Pachon's Sphygmometric Oscillometer.—This instrument, constructed on the aneroid principle, is much more convenient and sensitive and fully as accurate as the mercurial devices. In ordinary practice the cuff is applied at the wrist. The details of the technic accompany each instrument.

The Blood-Pressure under Normal Conditions.—The average systolic blood-pressure in the healthy young adult (20 to 25 years) when recumbent is 110 mm.; the diastolic 65 mm. and the pulse pressure 35 mm. The upper and lower systolic limits in health are about 145 mm. and 80 mm. respectively. The pressure in females is about 10 mm. lower. The pressure after fifty is higher, while the pressure in infants is about 80 mm. The normal pressure is influenced by physiological conditions. It is higher when erect than when recumbent, after eating; after mental and physical exertion providing they are not carried to the point of fatigue; the latter causes a fall in pressure. Sleep causes a slight fall in the systolic and a marked fall in the diastolic pressures.

The Blood-Pressure in Pathological States. High Blood-Pressure—Hypertension.—The highest recorded arterial pressures have occurred in acute compression of the brain, such as is caused by intracranial hemorrhage

or fracture of the base of the skull. Chronic arterial hypertension occurs in cases of intracranial pressure due to meningitis and tumor. As a general rule persons who become hemiplegic in consequence of thrombosis or hemorrhage have previously, if examined, manifested sclerosis of the peripheral vessels with hypertension. The apoplectic shock is attended with temporary hypotension.

Chronic Renal Disease.—Permanent high pressure is a conspicuous phenomenon in chronic interstitial nephritis. Systolic pressures of 200 mm. and more are common. Diastolic pressures are usually 60 to 80 mm. lower. The facts have great value in diagnosis. There are, however, cases of interstitial nephritis in which high arterial pressure does not occur. They are those with associated severe wasting diseases, those in which there is late cardiac insufficiency, and those that have reached the terminal stages of the disease. In chronic parenchymatous nephritis high tension also occurs, but is by no means so constant as in the interstitial form. In amyloid disease blood-pressure is inconstant, sometimes high, sometimes subnormal.

Uræmia.—The symptoms of this condition, especially in its chronic form, are associated with increased blood-pressure and become more marked as the tension rises, less marked as it falls. Persistent lower tension has followed improvement under treatment. A gradual fall has preceded death.

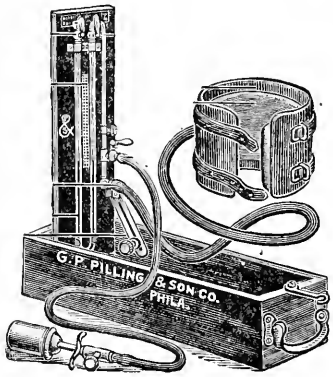


Fig. 70.—Faught mercurial sphygmomanometer.

Arteriosclerosis. — When the larger superficial arteries only are involved the blood-pressure is not markedly affected. Arteriosclerotic processes generally involving the smaller vessels are accompanied by increased blood-pressure, the systolic pressure being increased much more than the diastolic. There is accordingly a high pulse pressure. In this form of arteriosclerosis crises of vasoconstriction occur. Such vascular crises are met with also in tabes and chronic lead poisoning. To this group of paroxysmal constriction of the vessels must be referred angina pectoris, angina abdominis and intermittent claudication. During such attacks patients who ordinarily show hypotension may register very high arterial tension.

Diseases of the Heart.—In primary, uncomplicated cardiac insufficiency from myocardial changes high normal pressures appear to be the rule. When the cardiac insufficiency is due to failing compensatory hypertrophy in arteriosclerosis and renal disease, the blood-pressure is high. As the myocardium becomes feebler the arterial tension falls.

Valvular Disease.—In aortic insufficiency the sphygmomanometer, to use the words of Janeway, "gives a numerical value to the well-known pulsus celer, which expresses perfectly the mechanical effect of the lesion in the systemic arterial circulation." The systolic pressures are high, the diastolic pressures low. In combined aortic insufficiency and stenosis the blood-pressure determination is of value in indicating the preponderating lesion, a high degree of stenosis being accompanied by a proportionately

FIG. 71*a*.—Sanborn's sphygmomanometer. Auscultatory method.

FIG. 71*b*.—Sanborn's sphygmomanometer. Palpatory method.

FIG. 72.—Pachon's sphygmometric oscillometer.

lower systolic pressure. In associated aortic and mitral insufficiency, the degree of the latter defect may be estimated by the systolic as compared with the diastolic blood-pressure. In disease of the aortic valves the systolic pressure is frequently variable in the absence of obvious cause, while the diastolic pressure is more constant. Sphygmomanometric measurements are of less value in other forms of valvular disease. In aortic insufficiency there is sometimes a great difference between the maximal pressure in the arm and that in the leg, both being measured while the patient is in the recumbent posture. Normally these pressures are the same. Exophthalmic goitre shows as a rule normal pressure or hypotension. The relatively infrequent cases with hypertension usually show complicating implication of the heart, blood-vessels, or kidneys.

Low Blood-Pressure—Hypotension.—This condition is present in wasting diseases and cachectic states, various infections and toxæmias, especially when severe, profuse hemorrhage, collapse and shock, and terminal states—agonal hypotension.

Chronic Diseases.—Phthisis in its advanced stages and Addison's disease give low pressures. In the early stages of syphilis when there is fever and the condition is analogous to an acute infectious process, there is hypotension. Diabetes is apparently without direct influence upon the blood-pressure. When associated with arteriosclerosis or chronic renal disease it may show hypertension, and in advanced cases hypotension is common in consequence of emaciation and cardiac insufficiency. The secondary anæmias are attended by low blood-pressures; derangements of pressure in chlorosis are neither marked nor characteristic. Chronic bronchitis, emphysema and asthma are frequently attended with high arterial tension. Pleural effusions show hypertension which falls upon aspiration.

The Acute Infectious Febrile Diseases.—The type of this group, namely, enteric fever, shows with great constancy low pressure. Systematic observations at regular and frequent intervals have shown that hypotension is first apparent toward the end of the first or early in the second week and increases as the attack goes on. The daily oscillations are not significant. Crile's statistics, quoted by Janeway, are very suggestive. The mean pressure by weeks in all cases was, first week, 115 mm.; second week, 106 mm.; third week, 102 mm.; fourth week, 96 mm.; and fifth week, 98 mm. A gradually progressive fall indicates increasing failure of vasomotor tonus; a sudden fall, actual collapse or hemorrhage. A sharp rise in pressure attends the occurrence of perforation. These facts may be of great value in the differential diagnosis between collapse from hemorrhage or other cause in enteric fever and intestinal perforation. Continuous records are necessary. In the terminal stage of the consecutive peritonitis hypotension becomes extreme—agonal fall of pressure.

Pneumonia.—Uniform tendencies in blood-pressure have not been observed. Subnormal pressures are common; in severe cases the rule. A rapid fall may precede collapse or the fatal issue. When arterial pressure expressed in millimeters of mercury does not fall below the pulse-rate expressed in beats per minute, the fact may be taken as of excellent augury, while the converse is equally true (Gibson).

Diseases of the Nervous System.—In locomotor ataxia the lightning-pains are attended by a fall in blood-pressure; in the gastric crises

the pressure is greatly increased. Arteriosclerosis of the cerebral vessels may exist without similar changes in the general vascular system Blood-pressure estimations are therefore without value as indicating the existence of intracranial vascular lesions. When there is reason to suspect their presence, increased arterial pressure due to cardiac, vascular, or renal causes affords important data for prognosis and treatment. With high pressure there is danger of hemorrhage, with low pressure danger of thrombosis. Cerebral hemorrhage is attended by marked hypertension which continues to rise as the hemorrhage increases, and remains stationary or falls when the hemorrhage ceases. In uræmic coma the pressure is also greatly increased.

In epilepsy, owing to the difficulty of making observations during the attack, there is some uncertainty. During the attack there is said to be a sudden rise in the blood-pressure, followed by a rapid fall to normal as the paroxysm ceases. In coma following an attack of general convulsions the fact that in epilepsy the blood-pressure falls while in uræmia it remains high is of diagnostic importance. In tic douloureux there is a rise of pressure during the pain proportionate to the intensity of the attack. Insomnia may be associated with increased tension on the one hand or normal or diminished tension on the other. In the former condition the pressure falls during sleep. In hysteria and neurasthenia the pressures are variable. Some observers have observed high pressures in neurotic and excitable persons, but this condition is not constant.

Mental Diseases.—In melancholia the pressure is abnormally high and shows rises and falls corresponding to the intensity of the mental symptoms. In mania, on the other hand, the pressure tends to subnormal levels.

PERCUSSION.

Percussion in physical diagnosis is the art of striking or tapping upon the surface of the body in such a manner as to call forth sounds, from the nature of which conclusions are drawn as to the structure of the underlying parts.

This art was first described and systematically employed in the latter part of the eighteenth century by Auenbrugger, a physician of Gratz, who published his observations in a little book entitled Inventum Novum. The subject was widely brought to the attention of the profession by Corvisart in the beginning of the following century.

The practice of this method demands nice training both of the hands and ear in order to secure its best results. Careless and inexact methods yield not only unsatisfactory but also positively misleading results. It is especially true of percussion that they find it most useful who most clearly realize its limitations as an art in diagnosis. Unfortunately too many practitioners, otherwise well trained, fail to acquire proficiency in percussion and equally fail to appreciate its limitations.

Neither percussion nor auscultation requires the possession of much technical knowledge of acoustics nor a cultivated musical ear. It is, however, necessary to be able to discriminate differences in the character, intensity, and pitch of sounds.

The Theory of Percussion.—Reduced to its simplest terms the theory of percussion depends upon the differences in the vibrations produced by blows delivered upon structures which do not and those which do contain air, and in the latter case upon differences in the mechanical arrangement under which the air is present. There is nothing *a priori* in the matter. Our whole knowledge in regard to the signs elicited is the result of observation and experience. It has been found that direct percussion, that is, percussion without the intervention of a finger or other form of plexímeter, practised upon the thigh, which does not contain air, produces a minimum of sound which has a peculiar quality, technically described as dull. The interposition of a plexímeter increases the intensity of the sound and slightly alters its other acoustic properties. From this we infer that in percussion the vibrations of the plexímeter itself constitute a certain factor in the general result. It has further been found that percussion over the liver and spleen, organs which do not contain air, produces a similar dull sound, but that the quality of dulness is modified according to the force with which the act is performed. Upon light percussion over the spleen or centrally over the liver the dulness is much like that of the thigh, but upon powerful percussion over these organs the dull sound is modified, the quality of resonance being added. This fact in connection with others presently to be mentioned leads us to infer that by light percussion a limited region of the wall of the body is set into vibration, but that the area is extended by forcible percussion, so that the sound produced partakes of qualities due to the sound-producing mechanism of adjacent organs or structures, and that if we desire to obtain the percussion phenomena peculiar to an organ we must content ourselves with well-defined but light percussion of the surface overlying the viscus immediately in question. Experience amply confirms this inference. It has been further established that percussion over the distended bladder or a cyst, or any considerable collection of fluid, as a serofibrinous or purulent pleural effusion, produces a dull sound, and that there are degrees in the dulness just as we find differences upon light and heavy percussion over the spleen and liver, the sign having a certain quality of resonance at some parts of the border or edge of the effusion and wholly lacking resonance over the mass or base of the effusion. The recognition of these differences led to the very proper employment of such terms as *relatively dull*, *dull*, and *absolutely dull* or *flat*. To return to the liver and the flatness upon light percussion and the development of some degree of resonance upon forcible percussion especially near the borders of the dull area, we have attributed the latter to the vibrations of adjacent organs. Pursuing our investigations we find that as we proceed in lines upwards the sign changes somewhat abruptly from dull to a distinctly resonant sound, having qualities hereafter to be pointed out, which, with modifications of intensity and so on, but not of quality, is everywhere present over the chest where the surface or periphery of the lung comes into contact with the wall. For this reason the percussion sound elicited over the chest, and having the peculiar resonant quality spoken of, is known as pulmonary resonance, or briefly and technically as *clear*. Again, when we extend our percussion in lines proceeding downwards from the liver, we pass, under

normal circumstances, quite abruptly, about the margin of the ribs, to a region which yields upon percussion a note of high resonance having likewise peculiar qualities of its own, which, because of its being produced by a mechanism remotely analogous to that of a drum, is called *tympanitic.*

A very important fact in connection with these three fundamental qualities of the signs elicited upon percussion, namely, *dulness, clearness,* and *tympany,* is this, that they are constantly related to and dependent upon the absence or presence of air in the examined structures and upon the mode of arrangement of the air when it is present. The constant correspondence between the clinical and post-mortem percussion signs and the post-mortem conditions justifies us in formulating the following dicta: Upon percussion:

1. *Airless viscera and hollow viscera distended with fluid yield* **dulness, flatness.**

2. *The normal lungs contained in the chest under conditions of normal tension yield a* **clear note.**

3. *Air contained in hollow viscera, as the intestines, the walls of which are not tense, yield* **tympanitic resonance.**

These physical signs—namely, clearness, dulness, and tympany—are normal. The percussion sound clearness as such is always normal. It cannot be elicited anywhere save over the chest, and there is no condition of structures other than the lungs by which the physical arrangement essential to its production can be brought about. With dulness and tympany the case is different. The modifications or absence of clear or pulmonary resonance in regions normally occupied by the lungs constitute morbid physical signs. Dulness in regions normally clear or tympanitic and the extension of dulness beyond the limits of airless viscera constitute morbid physical signs, and this statement is also true of the presence of tympany in regions in which the physical conditions essential to its production do not normally exist.

The foregoing facts also warrant the following statements:
Upon percussion:

1. *There is no difference in the physical signs by which a distinction can be made between an airless viscus and a collection of fluid.*

2. *The signs do not enable us to determine the line of contact between two airless viscera or an airless viscus and a collection of fluid, or between collections of fluid separated by a membrane.*

Percussion is the application of an every-day art to diagnosis in medicine. The woodsman taps with his axe upon the trunk of a tree to learn whether or not it is hollow, the gauger upon the cask with his mallet to find the level of the wine, and the carpenter with his hammer upon the plastered wall to fix the position of a stud into which he can drive his nail.

The Technic of Percussion.—The patient may be examined in the recumbent, sitting, or erect posture. The outer clothing should be removed. The air contained in thick garments or in several layers of clothing seriously modifies the results of percussion. A single under-garment or a towel is preferable to the bare skin. The limbs should be symmetrically disposed and the muscles relaxed. Errors may arise from forcible percussion when the patient is resting upon a feather bed or very elastic

mattress. In general terms much display of energy on the part of the physician is to be avoided. It not only yields misleading results but it also alarms and may even hurt the patient.

Two methods are employed, immediate or direct, and mediate or indirect percussion.

Immediate or Direct Percussion.—The blow is struck directly upon the surface with the palm of the slightly flexed hand, or upon the clavicles or sternum with the tip of the second or third finger, or upon the abdomen with the dorsal surface—nail—of the second finger flicked off from the thumb as one flicks a crumb. The first two of these methods were originally employed. The last is a modern refinement.

With the Palm of the Hand.—The whole hand slightly flexed or the palmar surface of the fingers held closely together may be employed. The blow is delivered chiefly from the wrist, very slightly from the elbow, care being taken to avoid too much force and the over-production of noise. This method is available for a rapid preliminary survey and class demonstration of gross differences between the sides of the chest, or the upper and lower part of one side, especially posteriorly. It cannot often be employed satisfactorily in the examination of the abdomen. The objections to it are that it demands too much force and that the vibrations caused are too extensive. It lacks the nicety of good clinical work.

Direct finger percussion over the clavicles and sternum is often practised, but is here mentioned only to condemn it as mostly inexact, often misleading, and at best yielding results obtained much more satisfactorily by other methods. The results are unsatisfactory because of the elasticity and extensive vibrations of long and flat bones. The resonance produced is that of an elongated or very large plexímeter—so-called bone or osteal resonance, well illustrated upon percussion, in the same manner, of the head with the finger-tip. As there is no intracranial air, it is evident that the resonance is due to the vibrations communicated by the bone to the external air.

Direct Percussion or Finger-flicking. — In this procedure the skin should be bared. Very exact and satisfactory results may be obtained, especially in the examination of circumscribed regions in a thin-walled abdomen. It is by far the most satisfactory method of mapping out the limits of the splenic dulness.

Mediate or Indirect Percussion — Pleximetry. — The blow is delivered not directly upon the surface of the body but upon an interposed plate or disk of ivory or hard rubber—a plexímeter, literally, measurer of the blow. This instrument should be quite flat with rounded edges, about an inch and three-quarters in length and five-eighths of an inch in width, so that it may be closely applied to the surface in the intercostal spaces. There should be at each end a little flange or ear by which it is held in position. The percussing instrument or hammer is called a plexor, and consists of a suitable head of soft rubber, or metal tipped with soft rubber, and a light, stiff handle. The plexor of Wintrich has a handle or shaft nearly corresponding in length to a human hand from the wrist-joint to the first phalangeal joint and a head corresponding in length from the last named joint to the tips of the fingers. Instrumental pleximetry is much used among European physicians.

Finger Pleximetry—Finger Percussion.—This method is almost exclusively used by American physicians. A finger of the left hand is used as the pleximeter and the right hand as the plexor, the fingers being flexed as nearly as possible at a right angle at the first phalangeal joint to form the head of the hammer, and the hand from this joint to the wrist forming its handle or shaft. The blow is delivered from the wrist and not from the elbow, and the head of the plexor, made up of the last two phalanges, must fall at a right angle upon the dorsum of the middle or terminal phalanx of the finger used as the pleximeter, the palmar surface of which is closely applied to the part examined. It is scarcely necessary to add that in left-handed persons the fingers of the right hand are used as pleximeters and the left hand becomes the plexor.

The advantages of finger percussion are (a) that the soft palmar under-surface of the pleximeter can be closely applied to the part to be examined and the danger of a thin layer of air between them wholly avoided; (b) that the finger used as a pleximeter is also a palpating finger and receives sensory impressions concerning the firmness or elasticity of the underlying part which supplement the auditory impressions caused by the vibrations occasioned by the blow; (c) that the pleximeter is composed of tissues corresponding in physical composition with the wall of the body, which it protects from the blow without the interposition of an instrument of wholly different composition, and (d) that the instruments are always at hand.

Flicking percussion may also be intermediate, a finger of the left hand being used as the pleximeter.

Superficial and Deep Percussion.—These terms indicate in general the degree of force employed. In superficial percussion the blow is light and the vibrations are limited in extent and depth. This method is essential in the study of conditions in which the percussion signs involve limited areas, as in the heart and spleen, or in which we have to deal with thin wedges of tissues yielding different signs which overlie each other, as the lung surrounding the cardiac ventricles, or dipping down between the wall of the chest and the liver or the wedge-shaped anterior lower border of the liver occasionally seen. Superficial percussion enables us to determine the nature of the structure immediately beneath the surface, and is necessary where, by reason of the thinness and elasticity of the walls, wide areas of tissue are set into vibration by the blow, as in children and emaciated persons, and in elderly persons whose costal cartilages have undergone calcification. Only superficial percussion should be employed in the examination of the chest after recent hemorrhage.

Deep percussion excites vibrations in wide areas and to a considerable depth. It is employed where the chest walls are very muscular or fat and to ascertain the dulness or resonance of the deeper structures, as the actual limits of cardiac dulness, the upper border of liver dulness, pneumonic consolidation not reaching to the periphery of the lung, or a deep-seated aneurism. In the case of a wedge-shaped anterior lower border of the liver superficial percussion enables us to demonstrate the actual limits of dulness, while deep percussion, by acting upon the underlying intestine through the thin wedge of liver, yields a most misleading tympanitic resonance.

The following directions must be carefully observed:

1. Apply the second or ring finger of the left hand accurately and firmly but without undue pressure to the surface to be examined.

2. Raise the other fingers and palm from the surface to avoid muffling the vibrations. The finger used as the plexímeter only should at the moment be in contact with the surface.

3. Deliver a quick, rebounding blow, with the tip of the percussing finger or fingers perpendicularly upon the finger used as a plexímeter, upon the middle phalanx or the terminal phalanx above the nail. The quicker the rebound of the plexor the better and more significant the result.

4. Let the blow be delivered from the wrist held perfectly loose and not from the elbow. The force must be moderate and equal at corresponding points upon the two sides of the chest; lighter where the chest wall is thin, as in lean persons and in the infraclavicular and axillary and infra-axillary regions, and more forcible in the examination of the back of a very muscular man or the mammary regions of one who is fat.

5. The attitude of the patient is important. It must be easy and unconstrained. Rigid and fixed positions are to be avoided. Muscular tension modifies percussion resonance. The arms must be symmetrically arranged. In the examination of the anterior surface of the body let them lie loosely at the sides in the recumbent posture or hang relaxed if the patient is erect; in the examination of the back the patient should bend forward and gently fold his arms. I do not like the hands to be placed each upon the opposite shoulder, since it involves an undesirable degree of muscular tension; while in the examination of the lateral regions of the chest the hands should be placed together upon the top of the head with the fingers lightly interlocked.

6. The patient must breathe gently and regularly. If changes in the percussion signs upon full held inspiration and forced expiration are to be studied—respiratory percussion—give the necessary directions.

7. Perform percussion systematically and in a routine manner, examining corresponding parts upon the two sides of the chest above and below, anteriorly, laterally, and posteriorly, comparing and noting the signs at each step in the proceeding. Comparison and contrast are essential alike in percussion and auscultation. It is often useful to apply two or more fingers widely separated to the surface and lightly percuss one after the other. In this way the border-line between dulness and clearness or tympany can be defined and demonstrated with great exactness.

8. Deliver two or three percussion strokes and then examine the corresponding point upon the opposite side in the same manner. This manœuvre may be repeated as often as is necessary. Dexterity and close attention to the sounds render a wearisome prolongation of the examination unnecessary.

9. To determine the borders of areas of dulness, clearness, or tympany percuss in parallel or radiating lines and note the points in such lines at which the quality of the percussion signs changes. Repeated light percussion is often necessary. These points may be fixed by touches with the dermatographic pencil, which when joined by a line indicate the borders of the areas studied.

Practitioners gradually develop modifications of percussion methods to suit themselves. There are many different methods, but not every one of them is right. Those not based upon a knowledge of the principles upon which this method of physical diagnosis rests and those which are slovenly or careless are positively wrong. It is like playing a musical instrument. Knowledge, aptitude, and training are essential, and there are good performers, poor performers, and those who cannot play at all.

Sources of error especially to be avoided are:

1. Failure to apply the plexímeter accurately to the surface. A thin stratum of air modifies the result and may render it wholly misleading.

2. Applying the other fingers or the palm of the hand to the surface in such a manner as to dampen the vibrations and muffle the sound.

3. Awkwardness, slowness, and the use of too much force in delivering the blow. These may all be readily avoided if the percussion stroke is from the wrist as a centre of movement rather than the elbow.

4. A false attitude on the part of the patient. Many persons on being examined assume rigid and fixed postures with the muscles in tension and the arms in constrained positions.

5. Too much clothing, and setting the air contained in the pillow, bed, or mattress into vibration by powerful percussion.

6. A want of system in conducting the examination. More errors arise from carelessness than from ignorance.

THE SIGNS ELICITED UPON PERCUSSION.

The sounds differ among themselves, as already seen, as follows:

A. **Quality:** (1) Clear, (2) dull, and (3) tympanitic.

A structure containing no air yields upon percussion a minimum of sound due to vibrations in the surrounding air and is said to be completely dull or flat.

Changes in such a structure by which it becomes air-containing or the contiguity of air-containing structures modify the percussion sign, which acquires resonance, and the dulness is no longer flat or complete, but marked, and, as it is a question of degree, moderate or merely slight or relative.

The physical signs by which these modifications of flatness are brought to pass are (a) in the direction of the conditions which underlie tympany, namely, collections of air contained in spaces the walls of which are not too tense, as, for example, the intestines; and (b) in the direction of the arrangement of the air in the lung under normal conditions which involve a certain tension as to the vesicles and as to the whole lung within the thorax—clearness.

The terms used to designate (a) modifications of dulness in the direction of tympany are slight tympany, dull tympany, moderate tympany, and tympany.

Special modifications of tympany are cracked-pot resonance and amphoric resonance.

Cracked-pot Resonance: the Cracked-metal Sound. — This percussion sign requires for its development a rather forcible abrupt stroke while the mouth of the patient is open. The physical condition is an air-

containing cavity communicating freely with a bronchus and sufficiently near the surface of the chest to be compressed by the sudden blow. It may also be elicited, in the absence of cavity formation, in conditions in which by reason of yielding chest walls a certain amount of air contained in the lungs or in the pleural cavity is suddenly forced by strong percussion into the large bronchi. We may encounter the cracked-pot sound therefore in infants not suffering from disease of the lungs, especially when percussion is performed at the time of the full inspiration of crying, in pleurisy, above the level of an effusion, over lung relaxed by the pressure of a large pericardial effusion, sometimes in emphysema and in certain cases of pneumothorax. This modification of tympanitic resonance may be imitated by sharply percussing the cheek while the mouth is open or by striking the two hands held together against the knee in such a way as to cause a sound like that produced when coins are rattled in the hands. For this reason the cracked-pot sound is sometimes called the money-jingle sound. This sound is in many cases only to be heard when at the moment of percussion the patient's open mouth is turned directly toward the ear of the physician or when the patient holds the bell piece of a double stethoscope just in front of his open mouth. As sudden compression of the cavity is essential the blow must be of some force and as the walls of such a cavity are not always highly resilient the peculiar phenomenon in question is often produced only upon the first two or three strokes of percussion and sufficient time must elapse for the full redistention of the cavity before the cracked-pot sound can again be heard.

Amphoric or Metallic Resonance. — This sign has the quality characteristic of the sound produced by percussing a large vessel with a wide mouth—*amphora*, a jar. It is a ringing tympanitic sound and denotes a cavity of considerable size with firm elastic walls which do not vibrate in unison. The pitch varies with the shape and size of the cavity and the degree of tension of its walls. A closed cavity distended with air or gas under pressure so that its walls vibrate in unison yields dulness on percussion. Amphoric resonance frequently occurs without the cracked-pot quality, but the cracked-pot sound is usually also amphoric.

(b) Modifications of dulness in the direction of clearness are slight or relative dulness, impaired resonance, clearness.

But, leaving dulness altogether out of the question, we find that changes in the physical condition in the lung by which the normal or vital tension is relaxed frequently occur. This takes place, for example, in congestion, in œdema and atelectasis from compression, in both of which the residual air is diminished, and the normal, clear or vesicular resonance acquires the tympanitic quality to a varying degree—vesiculotympanitic resonance—and as the lesions upon which vesiculotympanitic resonance depends undergo resolution this sign is gradually replaced by the normal or clear resonance again. These changes can occur only in regions in which we normally find the clear or vesicular percussion resonance, namely, over the lungs. It has been demonstrated experimentally that the extreme distention of a hollow viscus, as a bladder, with air so that its opposite walls upon percussion do not vibrate independently, but as a whole, does away with the tympanitic sound, and causes it to be replaced by dulness.

B. **Volume or Intensity.**—This acoustic property is of minor importance in percussion. It depends upon the volume of air contained in the structures examined, the elasticity of the enclosing walls and energy of their vibrations, and the force of the blow. This term has reference to the loudness or degree of sonority of percussion sounds, which may be on the one hand so great as to obscure their value or on the other so faint as to be without any significance whatever. The duration of percussion sounds usually corresponds to their volume or intensity.

C. **Pitch.**—The distinction between sounds and musical tones must be borne in mind. In percussion we have to do with the former. Nevertheless the pitch of percussion sounds is of great importance. Pitch indicates in music the relative position of notes upon the scale and depends upon the frequency of the vibrations by which tones are produced. In physical diagnosis we find that large air-containing spaces with slight or moderate tension yield percussion resonance of low pitch, while small spaces with high tension yield resonance of higher pitch, and that the vibrations of the plexímeter upon the thigh or over an airless viscus yield a sound of slight intensity and high pitch.

Percussion over the abdomen reveals great variations in the pitch of sounds having the quality of tympany, as over the stomach and large and small intestines. These variations are of some value, but cannot be relied upon in mapping out the positions of those viscera. They serve a purpose in indicating the border-line between contiguous organs, as the stomach and transverse colon and coils of intestines under different degrees of tension.

The quality of a sound is that property which enables us to recognize it whenever heard without seeing the mechanism by which it is produced, as the sound of a bell, a drum, and so on; the volume or intensity of a sound relates to the energy and the mass of the material by which it is produced, as, for example, in great and little bells, the sound of which has the same quality and may have the same pitch while differing greatly in intensity or volume; the pitch depends upon the rapidity of the vibrations by which sound is produced, as in the long strings of the piano which produce low notes, and the short strings which send forth the high notes.

The Lung Reflex (Abrams). — It has been observed that local irritation of the skin of the chest as by cold or the application of mustard has been followed by the evidences of a temporary circumscribed emphysema of the underlying lung. These signs of dilatation of the air-vesicles have in some instances been confirmed by X-ray examination. Cabot has referred to this observation in explanation of the fact, well known to teachers of physical diagnosis, that the repeated demonstration of an area of moderate dulness, as, for example, in incipient tuberculosis, is followed by a modification of the percussion sign, which gradually becomes more resonant. The repeated percussion apparently acts as a local irritant. If the consolidation is dense and extensive this change cannot occur.

Respiratory Percussion.—Differences in the sound are noted upon quiet breathing and full held inspiration. The contrast between the two sides of the chest in slight consolidation, as in beginning phthisis or pleural thickening, is thus accentuated, the dulness upon the affected side remain-

ing the same, while the resonance upon the sound side is increased. This method is serviceable in determining the presence or absence of slight degrees of relative dulness, especially in the infraclavicular regions in incipient pulmonary tuberculosis.

Palpatory Percussion.—As has been pointed out in the general consideration of percussion, palpation is an essential though subordinate factor in finger percussion, which is gradually taking the place of other methods. Palpatory percussion is a method in which the attention is especially directed to the resistance and elasticity of the tissues over which the percussion is performed. It consists in the combined use of palpation and percussion in a modified form, and is applicable (a) to the determination of the outlines and boundaries of the solid viscera under various conditions, but especially to the study of the deep dulness of the heart both when the lungs are normal and when they are emphysematous; (b) to the examinations of solid organs of limited size surrounded by air-containing and resonant structures, as the spleen, and particularly when there is tympanitic distention of the abdomen; and (c) to the recognition of the extent and the horizontal levels of fluid exudates in the abdominal and thoracic cavities in different postures. The physical signs depend to a greater extent upon the sensation of resistance imparted to the percussion finger than upon the character of the sounds perceived, though both have value. Various methods have been described. Delicacy of touch, a light stroke, and a lingering rather than a momentary contact with the surface under examination are essential to success in all of them.

The direct methods of palpatory percussion are:

1. That of the Writer. — This consists in flicking the surface with the nail of the middle finger in the manner described under the caption direct or immediate percussion. This method is painless to the patient and yields very accurate results. The nail should strike the surface percussed flatly and linger for an instant.

2. Maguire's Method.—The palmar cushion of the tip of one finger is employed as the plexor. The stroke is not short and quick but prolonged and combined with a certain movement of pressure or palpation.

3. Method of Hein.—The first and middle fingers are employed, the tip of one resting upon the surface while the other, used as a plexor, delivers a light tap upon the adjacent surface, palpation and percussion being literally performed at the same time. The fingers are alternately used and the whole surface is gradually examined. Very accurate results may be obtained by this method.

The Indirect Methods Are: 1. The finger used as a pleximeter is struck lightly with the fingers of the other hand, which are slightly flexed in such a manner that the blow is delivered by the pulps rather than the extreme tips. The stroke is not sharp and rebounding, but prolonged and pushing, the so-called palpating stroke, and the percussing fingers remain a moment upon the plexor finger before the blow is repeated.

2. That of Ebstein. A glass pleximeter 4 centimetres in length and 1.3 centimetres in width, with a projecting bar 1.5 centimetres in height is used. This is held firmly in place while the finger held as in ordinary finger percussion delivers a gentle but pushing or pressing percussion stroke

upon the flat upper surface of the bar. The pleximeter devised by Sansom consists of a slender rod of square section having at one end attached at right angles a thin plate and at the other end a similar plate parallel to the first. The measurements are about the same as those of the glass pleximeter of Ebstein, but all the parts are made of hard rubber. In use the larger plate is applied to the surface of the chest and held in position by the tips of two fingers, one on each side of the rod. Percussion is then made upon the upper plate, the finger of the other hand being employed as a plexor. Greater attention is paid to the vibrations perceived by the fingers than to the sound. This special pleximeter enables the observer who has acquired skill in its use to recognize slight modifications of the vibrations produced by percussion and to map out more closely than by other methods, but not absolutely, the limits of the deep dulness of the heart and the great vessels.

Auscultatory Percussion.—The binaural stethoscope is applied to the surface of an organ, as the heart, liver, stomach, etc., and held in place by an assistant or the patient himself. Using finger pleximetry with very light strokes, percussion is performed in radiating lines towards or away from the stethoscope as a centre. Direct percussion with the finger-tips may be employed in case the observer himself is obliged to use one hand to hold the stethoscope in place, or a light stroking touch or scratching of the skin will serve the purpose. A stiff brush or the handle of a large tuning-fork in vibration may be used for this purpose. The sounds are greatly intensified and changes in their quality, volume, and pitch are readily appreciated. Of especial importance are the abrupt changes that take place as the line of percussion passes over the border of the organ over which the stethoscope is placed. The points at which the change occurs being marked and these points being joined by lines, an approximate outline of the organ is obtained. The observation must be controlled and errors eliminated by percussing in segments of widening circles and by the employment of the ordinary methods of percussion. This method is much more useful in the examination of the abdominal than of the thoracic viscera. It should be acquired by every student.

Percussion Signs in the Chest.

THE EXAMINATION OF THE NORMAL CHEST BY PERCUSSION.

Pulmonary Resonance.—The sounds elicited vary in different regions. The anterior and lateral surfaces are more resonant than the posterior by reason of the greater thickness of the walls of the last. The resonance in the former is known as normal, pulmonary, or vesicular. The portion of the apex of the lung above the clavicle yields a sound which acquires the tympanitic quality—vesiculotympanitic—as the trachea is approached. Some difficulty in the application of the finger or pleximeter renders percussion less satisfactory in this region than in other parts of the chest. Over the clavicle the sound has the peculiar quality known as osteal resonance; is dull towards the scapular extremity and acquires a distinctly tympanitic quality with heightened pitch at its sternal end of the bone.

In the infraclavicular region, that is, as far downward as the fourth rib, the pulmonary or vesicular resonance is characteristic. There is usually, however, a slight disparity in the two sides, the sound of the right being somewhat less resonant, shorter in duration, and of higher pitch than the left. The recognition of this fact is of cardinal importance. From the fourth rib downwards on the right side the resonance upon strong percussion is slightly diminished, owing to the presence of the dome of the right lobe of the liver. About the sixth rib the pulmonary resonance ceases. During full inspiration the liver is pushed downwards to the extent of an inch or more and the line of dulness is depressed to a corresponding degree.

On the left side the vesicular resonance is impaired by the presence of the heart between the fourth and sixth ribs and to the left as far as the mamillary or midclavicular line. The extent of this area is diminished under any conditions in which a larger wedge of the border of the lung is interposed between the wall of the chest and the heart, as upon deep inspiration and in those who have deep chests and voluminous lungs. At the base anteriorly the clear resonance passes into the tympanitic resonance of the fundus of the stomach—Traube's semilunar space; laterally into the dulness of the spleen.

In the lateral regions, axillary and infra-axillary, percussion yields vesicular resonance modified in the direction of higher pitch and diminished intensity towards the base of the chest by the presence of the liver on the right and the spleen on the left side.

Posteriorly the sound varies markedly according to the region percussed. The greater thickness of the muscles and the presence of the scapulæ are to be considered. The resonance is everywhere diminished as compared with the anterior and lateral regions. It has the quality of clearness but is muffled and merges into dulness over the scapulæ. The percussion sound is clear over the apices but usually slightly less so on the right side than on the left; and in the interscapular regions, which are widened when the patient bends forward and folds his arms. It is also clear from the angle of the scapula on each side to the base of the chest, namely, about the level of the tenth rib, where on the right side the liver dulness begins. On the left side the clear percussion sound may be found a little lower than upon the right; while the resonance upon deep percussion is somewhat diminished on the right by the convexity of the liver and on the left to a less extent by the spleen.

In children and emaciated persons the resonance in the back is often very good and percussion yields results scarcely less satisfactory than in the front of the chest.

Normal Tympanitic Areas in the Chest.—These are at the sternal ends of the clavicles, over the manubrium sterni and at the left base anteriorly. The first and second of these regions owe their tympanitic resonance to the proximity of the trachea and main bronchi and their osteal quality to the large proportion of bone entering into the wall of the chest. In elderly persons with calcification of the costal cartilages this osteal quality is widely present and when combined with tympany as is often the case greatly impairs the value of the percussion signs.

Percussion directly over the trachea at the episternal notch and that over the larynx, *i.e.*, over the plates of the thyroid cartilage, yields amphoric resonance. The normal tympany at the left base anteriorly is due to the presence of the fundus of the stomach when distended with air and the adjacent transverse colon. The curved upper border of this space, the convexity of which corresponds with the convexity of the diaphragm, is of special diagnostic value. The degree of distention of the stomach increases this curve, which is flattened or may even become concave in large left-sided pleural effusions. Traube's semilunar space is bounded to the right by the left lobe of the liver —dull; above by the lung—clear; to the left by the spleen —dull upon light percussion and is itself tympanitic, the tympany being continuous with that of the stomach and transverse colon. It often requires nice work in percussion to map out the border-line between the clear vesicular resonance which forms the upward limit of this space and the tympanitic resonance of the space itself.

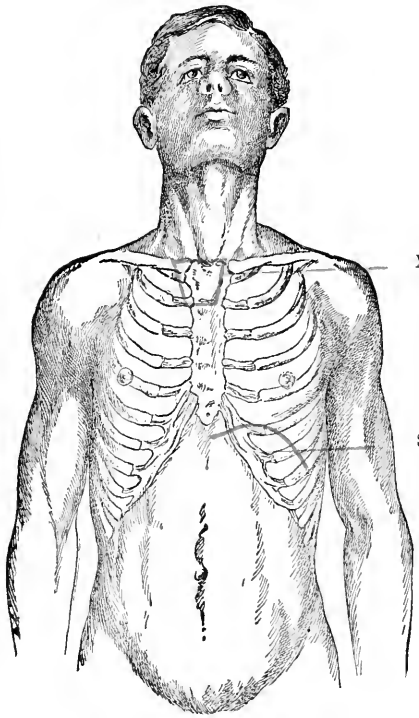


FIG. 73.—Normal tympany.

Dull Areas in the Normal Chest.—These are found to correspond to the scapulæ with their large muscular masses in the chest wall, the liver and spleen reaching up into the chest in the vault of the diaphragm and the heart within the chest itself. The scapular dulness has already been described. The liver dulness extends in the right midclavicular line from about the sixth rib to the border of the ribs and shifts downwards an inch or more on full inspiration; the dulness of the left lobe is continuous vertically with the cardiac dulness, from which it cannot be distinguished by ordinary percussion, although the border-line between them is sufficiently indicated for clinical purposes by the upper border of liver dulness on the right side and the position of the cardiac impulse on the left. In doubtful cases auscultatory percussion may be employed. The heart rests upon the central tendon of the diaphragm and the upper curvature of the liver fits into the vault of the diaphragm.

The area of the splenic dulness extends from the upper border of the ninth to the lower border of the eleventh rib and from a point slightly anterior to the midaxillary line backward towards the spine. It varies normally with the physiological changes in the size of the organ.

Cardiac Dulness.—The cardiac dulness is divided into the superficial and the deep.

Superficial.—The superficial cardiac dulness corresponds to that part of the organ constituted by the anterior surface of the right ventricle, which uncovered by lung lies in relation with the chest wall. It begins above about the level of the fourth costal cartilage and extends to the apex, being bounded below by the dulness of the left lobe of the liver and on the right by the resonance of the anterior border of the right lung at the middle line. This irregularly quadrilateral area varies in size according to the expansion of the borders of the lungs, especially the left. It is smaller upon inspiration than upon expiration and in active individuals with voluminous lungs than in sedentary persons with small and narrow chests. It yields upon superficial percussion a flat and upon deep percussion a dull note.

Deep.—The deep cardiac dulness corresponds to the borders of the heart itself beneath the overlapping margins of the lung and extends beyond the area of superficial dulness. Upon deep percussion over this area the note is dull, but the rounded receding surface of the heart renders a literally exact determination of its limits impracticable. Even the most skilful percussion yields only approximate results. The difficulties in determining the upper and right border of the heart by percussion are increased by the fact that the organ is covered in those regions not only by the borders of the lung but also by the sternum, which modifies to a high degree the percussion sound of the structures underlying it.

Enlargement of the heart gives rise to increase in the diameters of both these areas, the deep area of cardiac dulness being increased by the enlargement of the heart itself; the superficial area by the pushing aside of the margins of the lungs. The determination of the superficial area of cardiac dulness is a relatively easy matter, but the knowledge thus obtained relates rather to the position of the margins of the lungs than to the size of the heart; the determination of the deep area in so far as it is practicable would indicate the actual size of the heart, but the difficulties in reaching exact data are in many cases insuperable. For these reasons we cannot regard percussion as the best method of ascertaining the size of the heart. It has a value as a control method, but the position of the apex-beat, as determined by inspection, palpation, or auscultation, and the extent of the impulse, with associated clinical phenomena, constitute diagnostic criteria at once more convenient of application and far more precise.

The resonance of the normal chest is modified within narrow limits by a variety of conditions, among the more important of which are the following:

1. **Change of Posture.**—In the lateral decubitus the resonance of the lower lung is slightly less than that of the upper by reason of the greater amount of air in the latter. On exchanging the recumbent for the erect posture the pitch of the percussion sound is raised (Da Costa). If the patient turns upon the left side, the heart, under the influence of gravity, swings outwards towards the left axilla, with a corresponding change in the position of the apex and the cardiac dulness.

2. **Respiration.**—The general resonance of the chest is greater upon full held inspiration than on quiet breathing simply because of the increase

of air within its cavity. This increase of resonance may be noted on quiet respiration after great muscular exertion, which is accompanied by a temporary physiological distention of the vesicular structure of the lungs.

The increase in the volume of the lungs upon full inspiration not only augments the resonance but also extends its borders in certain directions, especially over the heart so that the superficial area of cardiac dulness is diminished, and at the base of the chest so that the liver and spleen are carried downwards with the descending diaphragm, and areas at the base, dull on expiration or quiet breathing, yield a clear note. This respiratory excursus of the lower margin of the lungs is observed posteriorly as well as anteriorly, but not to the same extent. It varies in different individuals in health just as the inspiratory expansion varies and is diminished by the presence of pleural adhesions.

3. **Gaseous Distention of the Stomach and Colon.**—This condition may displace the upper crescentic convexity of Traube's half-moon-shaped space and cause tympanitic resonance in the lower part of the left chest or impart a tympanitic quality to the vesicular resonance—vesiculo-tympanitic resonance. It may also to some extent displace the diaphragm upwards, thus causing the lower margins of the lungs to assume a position slightly higher than normal with a corresponding upward displacement of the limit of pulmonary resonance.

Age.—In children the lungs are relatively small and the dull areas of the heart and liver correspondingly greater. In old age the borders of the lungs are usually emphysematous, even in persons otherwise in normal condition. Hence the area of superficial cardiac dulness is encroached upon and the upper border of liver dulness is slightly lower than at earlier periods of life. Under this circumstance the vesicular resonance acquires a faintly tympanitic quality.

The Condition of the Chest Wall.—The obvious part in this respect played by great muscular development and obesity has already been spoken of. There are persons in whom percussion on account of these obstacles yields negative results. Œdema of the chest wall is also an important obstacle. Highly developed mammæ likewise interfere with the application of this method of examination; so also do the tenderness of inflammation of the chest wall and hyperæsthesia.

PERCUSSION IN DISEASE OF THE THORACIC ORGANS.

Percussion in the different regions of the normal thorax yields (1) vesicular resonance, the sign of normal lung tissue under normal intra-thoracic tension; (2) diminished resonance or dulness over the scapulæ and the area of deep cardiac dulness; (3) absence of resonance or flatness over the lower ribs on the right side anteriorly; (4) vesiculotympanitic resonance towards the base of the chest anteriorly on the left; (5) tympanitic resonance over Traube's semi-space and over the manubrium and the sternal ends of the clavicles; (6) amphoric resonance over the trachea and cracked-pot resonance sometimes in the crying infant. While these sounds are normal when obtained in the particular regions of the chest above indicated, they become abnormal or morbid signs in other positions.

Thus vesicular resonance in the area of superficial cardiac dulness may indicate dextrocardia or some other form of malposition of the heart; diminished resonance or dulness in the infraclavicular or mammary regions may be significant of tuberculous infiltration or at the bases of bronchopneumonia; absence of resonance or flatness over a large area on either side which is normally clear denotes pleural effusion, a tumor, or some other airless condition; vesiculotympanitic resonance is the sign of a moderate degree of atelectasis due to compression of the lung and of emphysema; when of high degree it constitutes the percussion sign known as skodaic resonance; tympanitic resonance elsewhere than normal is the sign of a cavity, pneumothorax, or pneumopericardium; and amphoric resonance save over the trachea and the cracked-pot sound except in crying children must in all instances be looked upon as pathological conditions.

The changes which modify the normal resonance affect (a) the borders of the lungs; (b) the structure and tension of the lungs, and (c) adjacent organs. They may be general, unilateral, or local.

Changes in the Relation of the Borders of the Lung to the Wall of the Thorax.

The Apices.—The lungs normally reach about an inch and a half to two inches above the clavicles, the right apex being usually somewhat higher than the left. Normal pulmonary resonance is obtained therefore in both retroclavicular spaces. If absent in one or both and especially when replaced by dulness there is consolidation and retraction of the apex or apices. This sign is significant of tuberculous disease, fibroid phthisis, or local adhesive pleurisy. It is often stated that bulging of the retroclavicular space with tympanitic resonance occurs in emphysema. This is not always true. Owing to the skeletal changes in the thorax in emphysema of high grade there is usually retraction of the spaces immediately above and below the clavicles. Transient prominence with vesiculotympanitic resonance occurs in the acute emphysema of asthma and pertussis.

The Anterior Borders.—The resonance is marked by the osteal quality of the percussion sound over the sternum and the signs are uncertain. Below the level of the fourth costal cartilage the border of the left lung sweeps downward and to the left, and is readily made out by percussion, forming the upper and left lateral boundary of the area of superficial cardiac dulness. Below the clinical apex of the heart and between the anatomical apex and the chest wall a tongue-like projection of the anterior border of the lower lobe called the lingula gives rise to a clear percussion sound over a limited wedge-shaped space. The anterior border of the left lung is pushed aside by an hypertrophied heart or large pericardial effusion so that the area of superficial cardiac dulness is increased. An increase in diameter is, however, much more frequently due to diminution in the volume of the lung as in tuberculous or fibroid disease and consequent retraction of its borders. In substantive chronic emphysema and the acute emphysema of asthma and whooping-cough the left border of the lung is advanced and in extreme cases to such an extent as to obliterate the area of superficial cardiac dulness.

The Lower Borders.—Due allowance must be made for the changes caused by posture, exertion, age, etc. (p. 141). The borders are lowered

in pathological conditions in which the volume of the lungs is increased and they occupy a position higher than normal when it is diminished.

In advanced cases of emphysema the lower border of the lung as marked by the transition to liver dulness on the right side and to tympany on the left, may reach the ninth rib and a corresponding level in the lateral and posterior regions. The respiratory excursus of the border is very limited in this disease. It is likewise much restricted by old pleural adhesions. Permanent upward displacement with restricted respiratory movement is a sign of tuberculous or fibroid shrinking, chronic bronchopneumonia, or pulmonary collapse. The lung border may be pushed up by a distended abdomen, or drawn up by its own elasticity in paralysis of the diaphragm. It is also displaced upward and rendered immovable by fluid or air in the pleural cavity—hydro-pyo-hæmo-pneumothorax.

Impaired Resonance; Dulness; Flatness.—The resonance is diminished in proportion as the amount of air is decreased in the part percussed. It is modified according to the changes in the physical structure of the spaces containing the air caused by the lesions of disease. Consolidation of the lung from exudate within its substance, compression, infarct, collapse, renders the percussion sound over the affected area less resonant in proportion to the extent of the lesion. In disseminated lesions, as those of the common forms of ordinary or tuberculous bronchopneumonia, there is usually a local compensatory emphysema which modifies the dulness. The association of heightened pitch and diminished volume with diminished pulmonary resonance must always be borne in mind. In many instances the well-trained ear will recognize a change in the pitch of the percussion sign before alteration in its quality. The sensation of increased resistance—loss of elasticity—which is experienced by the plexímeter finger in fluid exudates and dense consolidations is also to be remembered.

Impaired resonance is a sign of beginning or disseminated tubercle, bronchopneumonia, early croupous pneumonia, small effusions, thickened pleura, gangrene of limited extent, and small abscesses or tumors. Dulness is present when the lesions of the above conditions are close set or extended.

Flatness when no Air is Present.—The percussion sound is dull over the complete consolidation of croupous pneumonia involving a lobe or an entire lung because some air yet remains in the large and middle-sized bronchial tubes; it is flat over a large effusion because the lung with its compressed vesicles and with it the air-containing bronchi are pushed wholly away. The presence of circumscribed consolidations, especially when not directly beneath the chest wall, cannot be recognized by percussion. Their only sign may be a slight elevation of the pitch. Hence central pneumonias and deep-seated aneurisms are frequently overlooked. An effusion into the pleura of serum, pus, or blood which does not reach 500 to 750 cubic centimetres in volume does not often yield definite physical signs upon percussion, and a pericardial effusion of half this amount may escape detection. In pneumothorax when the bronchopulmonary fistula has closed and the air is present under a high degree of tension, the percussion note over the greater part of the affected side may be dull.

Impaired resonance over the apex or upper lobe of one lung with normal resonance elsewhere is commonly significant of tuberculosis. It

may, however, be caused by an apex pneumonia or gangrene. Dense pleural thickening is also a cause of dulness in this region. Slight impairment of resonance in this region which passes away upon repeated deep inspiration or prolonged percussion may simply indicate habitual deficient respiratory expansion of the lungs.

Dulness at the base of the chest, always more pronounced and significant posteriorly, may be the sign of pneumonia, œdema, hypostatic congestion, atelectasis, or pleural effusion or thickening. Less commonly it stands for infarct, abscess, gangrene, tuberculosis, or tumor.

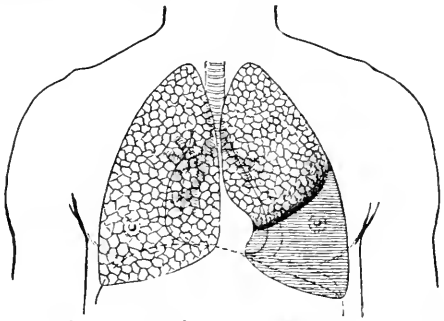


FIG. 74.—Pleural effusion, left side, showing degree of displacement of heart and of obliteration of Traube's semilunar space.

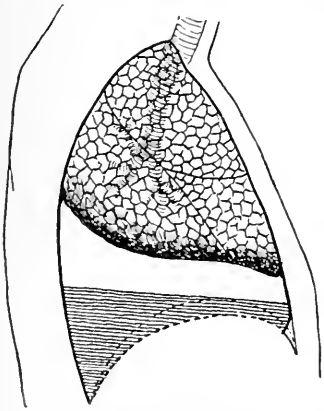


FIG. 75*a*.—Pneumohydrothorax—erect posture.

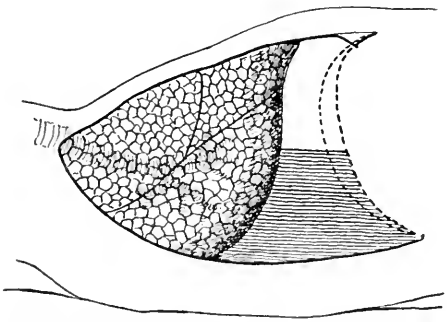


FIG. 75*b*.—Pneumohydrothorax—dorsal decubitus.

Flattening of the convexity of Traube's semilunar space is a sign of moderate pleural effusion; marked depression, with a concave upper line, occurs in massive effusion.

Vesiculotympanitic resonance of woodeny quality is significant of extensive fibroid changes in the lung.

Dulness at one or the other base, the upper line shifting quickly upon change in posture, is characteristic of pneumohydrothorax. The upper line of small pleural effusions shifts much more slowly and that of large effusions scarcely at all save in prolonged and decided change of posture. It is to be remembered that a pleural effusion which develops insidiously while the patient is up and about occupies the lower part of the chest and causes dulness at the base anteriorly, while one that accumulates in a bed-

ridden patient may cause extensive dulness posteriorly and reveal itself anteriorly merely by skodaic resonance. The significance of dulness in the interscapular region is often obscure. It may be a sign of pulmonary collapse or great enlargement of the bronchial glands. In the latter case there is also dulness instead of osteal tympany over the lower cervical vertebræ. Dulness or flatness in the left suprascapular or particularly in the left interscapular space may be caused by the presence of an aneurism of the descending aorta.

Increased Resonance—Hyperresonance—Vesiculotympanitic Resonance—Tympany.—Solidification of lung tissue changes its percussion note to dulness. An increase in the amount of air causes an increase of resonance, but does not necessarily change the quality of the note, which retains its clearness alike in shallow-chested and in deep-chested individuals and in forced expiration and in full held inspiration. In truth the change from the clear to the tympanitic percussion note very frequently accompanies a reduction in the amount of air contained in the portion of the lung under examination. The resonance has a tympanitic quality in extreme dilatation of the air-cells, as emphysema, in deep congestion, œdema, the pressure atelectasis overlying an effusion or adjacent to a tumor, and that part of the lung which is the seat of collateral fluxion in pneumonia; it may be exquisitely tympanitic in any of these conditions.

Clearness is replaced by tympany over portions of the lung which have broken down with the formation of cavities, provided that the cavities contain air; when they are filled with fluid the percussion sound is dull.

The note is tympanitic in pneumothorax and in the rare instances of pneumopericardium that occur. But when the cavity in pneumothorax is closed and the air is present under high tension the note becomes dull.

The tympanitic percussion sound may be due to extrapulmonary conditions. We have seen that percussion of the parts immediately over the trachea and main bronchi yields resonance having this quality. In the same manner inexpert percussion over a consolidated lung may yield a tympanitic sound due to the air in the trachea and large bronchi on the one hand or to the air in the stomach and intestines on the other.

Finally, the bases of the chest posteriorly in crying infants in health often yield a tympanitic sound, and that sound, as has been pointed out, sometimes has the cracked-pot quality.

When we come to review the physical conditions present under the foregoing circumstances, we are impressed with the fact that, whether directly or by conduction, the vibrations produced by percussion act upon air-containing structures which do not fulfil the requirements of the clear percussion sound, namely, air contained in elastic vesicles under physiological tension within the chest. On the contrary, they present, completely or in a modified manner, the very conditions necessary to the tympanitic percussion sound, namely, air in spaces, the walls of which are not under any great degree of tension.

In emphysema we recognize as an essential lesion that nutritive change in the alveolar walls which interferes with expiratory contraction; even in local or compensatory emphysema there is some degree of impairment of contractility from vesicular overdistention. In congestion and œdema

the volume of air in the chest is decreased as the quantity of blood is increased or serum is present and the normal tension diminished to a corresponding degree. In compression of the lung the air is squeezed out of the atelectic portion as the water out of a sponge and the vesicular tension is done away with altogether. In collateral fluxion the condition is the same as in congestion from other causes. In the foregoing conditions the percussion sound varies from vesiculotympanitic resonance—mere hyperresonance—to an exquisite tympany. In air-containing cavities within the lungs or in the pleural space and in the case of the tracheobronchial or gastro-intestinal tympanitic sound we have to do with the conditions essential to this phenomenon and the quality is unmixed and constant.

The pitch of the tympanitic sound varies with the degree of tension of the air within the cavity, becoming higher—dull tympany—as the tension increases, and with the relative width of the opening with which the cavity communicates with the air, the wider the opening the higher the pitch. The student may demonstrate these facts by percussing his distended cheeks under progressive degrees of tension with his mouth closed and with his mouth opened progressively to the full extent.

1. Vesiculotympanitic resonance on both sides of the chest is significant of emphysema, which may be acute as in asthma or pertussis, or chronic as in pseudohypertrophic emphysema. The degree of tympany varies with the grade of the disease. In extreme cases the percussion sound becomes high in pitch, small in volume, and short in duration—dull tympany.

2. The percussion note is hyperresonant and has the tympanitic quality over the sound side in the vicarious respiration such as occurs in extensive disease of the lung, massive pleural effusion, or large tumor of the opposite side.

3. An exquisite tympanitic sound, often partaking of the amphoric quality, is present over the affected side in pneumothorax. With extreme intrapleural tension the sound becomes less resonant—dull tympany or flat tympany—or may become quite dull.

4. Local tympanitic percussion resonance is a constant sign of pressure atelectasis. It is present at the level of pleural effusions, above towards the axilla and at the base posteriorly on the left side in massive pericardial effusions, and surrounds the dulness caused by pleural and pulmonary tumors. In old cases, as the atelectasis becomes complete, the tympanitic resonance is replaced by dulness.

5. Tympanitic resonance in one or both infraclavicular spaces associated with dulness at the base of the chest may be a sign of pleural effusion, pneumonia of the lower lobe, infarct, abscess, gangrene, or, especially when bilateral, of œdema.

6. Tympanitic resonance at the sternoclavicular articulation and below it with dulness at the apex is usually conducted tracheobronchial resonance. It is encountered in tuberculosis and apex-pneumonia.

7. Circumscribed tympanitic percussion resonance is the sign of a cavity which may be tuberculous, bronchiectatic, or the result of abscess or gangrene. The nearer the cavity lies to the surface the better defined the tympany. Consolidation of the intervening lung tissue acts in the same way. In tuberculosis a cluster of small communicating cavities is often

present at the apex. Single small cavities even when they are superficial, and deeply seated cavities even when of moderate size, do not yield a tympanitic percussion sound.

8. Subdiaphragmatic tympanitic resonance when the distention is extreme may be elicited by percussion in the anterior axillary line on the left side as high as the third interspace.

Amphoric Resonance.—This is the sign of a cavity of large size or, when very extensive, of pneumothorax. The cracked-pot sound is usually the sign of a cavity of some size with compressible walls and communicating freely with a bronchus. The essential physical requirement is that the walls should be freely compressible so that there may be a free outrush of air at the moment of the percussion stroke.

The following percussion phenomena described in the text-books are of greater clinical interest than practical value. They are very rarely brought out in a manner that amounts to a demonstration.

1. Wintrich's Sign.—The tympanitic percussion sound is higher in pitch upon opening the mouth and lower when it is closed. The patient should open his mouth, protrude his tongue, and breathe quietly. This phenomenon is occasionally, but by no means in the majority of instances, observed in large cavities of the lung or pneumothorax with wide communication with a bronchus.

2. Interrupted Wintrich's Sign.—If the foregoing sign is exclusively present in the sitting posture, it is evidence of a cavity containing fluid which in one posture occludes and in the other leaves open the communication with the bronchus. Under these circumstances gurgling or the râle of cavities is usually present.

3. Gerhardt's Sign. — The pitch changes with change of posture, usually becoming higher in the erect posture but scarcely ever becoming higher in the recumbent position. The alterations in pitch are attributed to the changes in the shape of the cavity caused by the gravitation of the fluid to its lowest part.

4. Friedreich's Sign. — The tympanitic resonance over a cavity communicating with a bronchus is higher in pitch upon inspiration than upon expiration. The change in pitch is always slight and often too slight to be of value in diagnosis. The inspiratory rise is attributed to the widely open glottis and the increased tension of the air in the cavity.

5. Biermer's Sign. — In pneumohydrothorax the tympanitic percussion note is lower in the recumbent than the erect posture, the change being due to alterations in the shape and relative diameters of the air space caused by the gravitation of the fluid. The underlying principle in Gerhardt's sign and Biermer's sign is the same.

Coin Percussion—Coin Test—Anvil Test.—Auscultation is performed upon the chest while an assistant percusses at a point diametrically opposite upon the front or back as the case may be, using a coin laid flat upon the surface as a pleximeter and another as the plexor, striking with its edge. The coins should be of some weight, as fifty-cent pieces or silver dollars. If pneumothorax be present the peculiar auscultatory sign consists of a clear metallic, ringing, bell-like note. Control observations may be made upon the opposite side and over the compressed lung. This sign does not occur over very large cavities—*vomicæ*.

PERCUSSION IN DISEASE OF THE HEART.

The normal superficial and deep cardiac dulness and the method of determining them have been discussed in a foregoing section. By this method of physical diagnosis we ascertain approximately the size, shape, and position of the heart, the relation of the anterior borders of the lungs, especially upon the left side, to it, and the presence of pericardial effusion when it is of sufficient amount. The area of superficial cardiac dulness is ascertained by light percussion in the parasternal line from above downwards until dulness with a distinct sense of resistance is reached, usually about the level of the fourth rib or its lower border. This point is in a transverse or oblique line extending downward and outward from midsternum. Next percuss over the lung upon the right side about the level of the fifth rib and in a transverse line across the sternum to the left. About or just beyond the middle line the sound again becomes dull and the resistance increased. This marks the limit of the anterior border of the right lung. Continuing to percuss in the same line and lightly as before, we reach a point at which the sound again becomes clear and which corresponds to the anterior border of the left lung at the level named. This determines the transverse diameter of the superficial area of cardiac dulness. The left lower angle corresponds to the apex and can be determined by palpation. The lower border is bounded by a line prolonged from the upper border of liver dulness to the apex of the heart.

The area of deep cardiac dulness may be roughly estimated by percussion in corresponding parallel lines from the parasternal line on the right side across the chest to the left and from above downward upon the left side along the sternal border, the parasternal line and the mamillary line. The base line is the same in both areas. The first modification of clear pulmonary resonance as we approach the heart may be accepted as a sign indicating the outline of that organ.

SIGNIFICANCE OF VARIATIONS IN THE CARDIAC DULNESS.

Alterations in the size of the superficial area, as has been heretofore stated, are usually signs of pulmonary rather than of cardiac disease. They correspond to increase as in emphysema, or decrease as in phthisis in the volume of the lung. This area together with the deep area undergoes changes, however, with changes in the volume of the heart.

Increase of Cardiac Dulness.—When the enlargement, as determined by percussion and other methods, extends chiefly to the left and downward, the longest diameter being oblique from above downward and to the left, it is the sign of hypertrophy and dilatation of the left ventricle. When the enlargement is to the right, with an extension of the dulness in the third and fourth intercostal spaces at the right border of the sternum and a rounded blunt apex, the longest diameter being transverse, it is a sign of hypertrophy and dilatation of the right ventricle and auricle. Enlargement both to left and right indicates hypertrophy and dilatation of both ventricles, the dilatation under these circumstances being almost always in excess of the hypertrophy.

Enlargement of the boundaries of precordial dulness to the left, right, and upwards may indicate the presence of a pericardial effusion. The dulness is marked and its outline is pyramidal or pear-shaped, the smaller end being directed upward. The increase in dulness may be first observed in the angle formed by the right border of the cardiac and the upper border of the liver dulness, which becomes at first rounded and then obliterated. The dulness may extend to the second interspace or higher and is sharply defined at its borders. In pericardial effusions of considerable size the compressed left lung yields a vesiculotympanitic or tympanitic resonance —dull tympany; skodaic resonance. The apex-beat may be felt or located by the intensity of the first sound within the borders of dulness. Percussion alone will not always enable us to make a differential diagnosis between a moderate pericardial effusion and extreme dilatation of the heart.

Under normal conditions the impairment of resonance due to the presence of the aorta and pulmonary artery does not extend beyond the manubrium on either side. When it can be made out upon the right side in the first and second interspaces, or in the notch of the sternum, it is usually a sign of dilatation of the aorta or of aneurism of the ascending or transverse portion of the arch. Sometimes it indicates the presence of a mediastinal tumor.

Decrease in the area of cardiac dulness is mostly caused by pulmonary emphysema, by which the heart is covered more completely by the lung and displaced towards the middle of the thorax. The heart like other muscle masses undergoes atrophic changes in acute and chronic wasting diseases, as enteric fever and consumption, with a corresponding diminution in the area of cardiac dulness. This area is diminished in extent, altered in outline, and in extreme cases rendered wholly unrecognizable by percussion in left pneumothorax, pneumopericardium, and emphysema of the mediastinum, such as follows trauma and occasionally occurs in fatal cases of pertussis or after tracheotomy. Extreme meteorism and great distention of the stomach by gas may cause like effects. Under these conditions the modified pulmonary resonance of the deep area and the dulness of the superficial area are replaced by a tympanitic note.

Dislocation of the Cardiac Dulness.—The heart is a very movable organ. The shifting which the apex undergoes upon changes of the posture of the body has already been described. The heart is displaced upwards by great meteorism, ascites, a massive abdominal tumor, or pregnancy; to the left by pleural effusion, pneumothorax, or tumor on the right side; to the right by similar conditions upon the left side. Great enlargement of the right lobe of the liver likewise displaces the heart to the left. Contraction of the lung with adherent pleurisy displaces the heart by traction toward the affected side, as in old pleural effusions that have undergone resorption or been relieved by operation, and in cirrhosis of the lung. Aneurismal or cancerous tumors and diaphragmatic hernia are among the rarer causes of displacement of the heart. Practically speaking, displacements of the heart are the result of diseases of the pleura or lungs. The greater part of the heart and its apex may lie to the right of the median line with or without general transposition of the viscera. Under all these conditions except the last, there are such modifications of the percussion

signs relating to the heart as to render that method of physical diagnosis still more unsatisfactory and frequently wholly useless in determining the boundaries of the cardiac dulness, valuable as it remains in the diagnosis of the primary disease. The position of the apex as located by palpation and auscultation constitutes the most reliable evidence of the region occupied by the displaced heart. The diagnosis of congenital displacement of the heart should in no instance be made until all other causes capable of producing such displacement have been shown to be absent.

Percussion in the Examination of the Abdomen.

This method is far less valuable than palpation in the diagnosis of abdominal diseases. It has, however, much usefulness for direct examination and is particularly important in controlling the results obtained by the other methods of physical examination.

The Technic.—The general directions are the same as in the technic of palpation. Direct percussion except flicking percussion cannot be employed, owing to the sensitiveness of the surface and the elasticity of the walls of the abdomen. As in palpation the patient must be examined in various postures, and it is frequently necessary to distend the stomach or colon with air or water. Auscultatory percussion is of service in determining the boundary lines between contiguous viscera, whether they be solid or air containing.

PERCUSSION OF THE ABDOMEN IN HEALTH.

The upper limits of hepatic and splenic dulness are determined by vesicular resonance. With this exception the signs relate to dulness and tympanitic resonance and their modifications.

The normal dull areas in the abdomen are:

(a) **Hepatic.**—The upper border of dulness begins about the level of the sixth rib in the midclavicular line. Its lower border nearly corresponds with the arch of the ribs. This area of dulness shifts with the respiratory movements about two fingers' breadth on quiet and slightly more upon deep breathing. The dulness extends upon corresponding lines in the epigastric zone and its respiratory excursus is less at the back than in front. The dulness of the left lobe extends to the left of the median line and is continuous vertically with that of the heart.

(b) **Splenic.**—The dull area of the spleen occupies the space between the ninth and eleventh ribs, its anterior border being slightly in advance of the midaxillary line. Its respiratory excursus is slightly less than that of the liver. The observation must in all cases be confirmed by palpation.

The above are constant in health.

Dull areas that are not constant also occur under physiological conditions. These are:

(c) **The Distended Bladder.**—The dull area is situated in the suprapubic region in the median line. It may extend half-way to the umbilicus. It is oval and symmetrical in outline and disappears upon micturition or catheterization.

(d) **A Distended Stomach.**—A hearty meal or the large ingestion of fluid will cause an area of dulness in the epigastrium which disappears in the course of digestion. The sharp contrast between the lower border of the dulness and the tympanitic resonance of the transverse colon indicates the position of the greater curvature of the stomach.

(e) **Fecal Masses in the Colon.**—In persons of sedentary habits it is not uncommon to find areas of dulness corresponding to the course of the colon, and especially to the left end of its transverse portion, which disappear upon brisk and repeated purgation.

(f) **Pregnancy.** — The oval area of dulness gradually developing upward from the pelvis, always central, at first symmetrical, later deflected somewhat laterally, is suggestive. The diagnosis of this physiological condition under ambiguous circumstances or in a doubtful case must be a guarded one.

With the above exceptions the percussion resonance of the abdomen is tympanitic. Its pitch varies with the dimensions of the particular space and the tension of the contained air, being relatively high as the space is small and the tension great. The stomach and colon yield therefore a percussion note of lower pitch than the small intestines. The structure and functions of these organs are, however, such as to cause great variations in the size, tension, and relation of their various parts, and lessen the value of the signs obtained by this method of examination. The percussion signs are furthermore greatly modified by the thickness of the abdominal walls and their general state as to tension and relaxation.

PERCUSSION IN DISEASE OF THE ABDOMINAL ORGANS.

Under ordinary circumstances except as above stated the abdomen in health is everywhere tympanitic beyond the borders of the liver and spleen. Persistent dulness is significant of morbid conditions. It may be general or local, continuous with the dulness of the liver or spleen or separated from them, fixed or shifting.

General Dulness of the Abdomen.—The retracted abdomen seen in the wasting diseases and in œsophageal and pyloric carcinoma, cholera, and the pernicious vomiting of pregnancy is usually dull upon percussion. The areas of tympanitic resonance are limited in extent and of irregular distribution. This is especially true of the scaphoid abdomen so often observed in meningitis, tumor of the brain, and lead colic. The bowels are empty of air and collapsed.

The general distention due to fat in the walls and intra-abdominal fat, fluid within the peritoneal cavity, or abdominal tumor yields dulness upon percussion. The bowels contain air but under conditions which modify the results of percussion. In the case of an excess of fat in the walls the force of the blow is not transmitted to the underlying gut; in excessive omental fat the same is true. Fluid accumulates in the dependent parts, displacing the coils of intestine, which float upon the surface, and yields dulness upon percussion at the lower levels with tympany above shifting with change of posture, the line between them tending to maintain its correspondence with the plane of the horizon. Thus, in the recumbent

posture there is general dulness save in a limited oval region around the umbilicus, over which there is tympanitic resonance; in the erect posture the resonance of this region is replaced by dulness while there may be demonstrated a broad line of tympanitic resonance in the epigastric zone, previously dull; in the lateral postures the area of resonance seeks the upper spaces and shifts alternately as the patient turns from side to side. The fluid commonly gravitates slowly from region to region and a few moments must be permitted to elapse before the change of note can be demonstrated. Large monocysts, as of the pancreas or ovary, also yield fluctuation and general dulness, but the intestines do not float at the highest level, being,

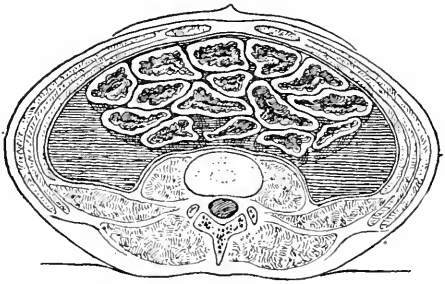


FIG. 76.—Free fluid in abdominal cavity—dorsal decubitus—flatness in flanks and tympany over supernatant coils of intestines.

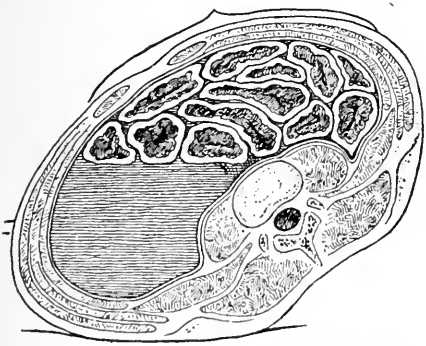


FIG. 77.—Free fluid in peritoneal cavity—lateral decubitus—flatness in dependent side and tympany above.

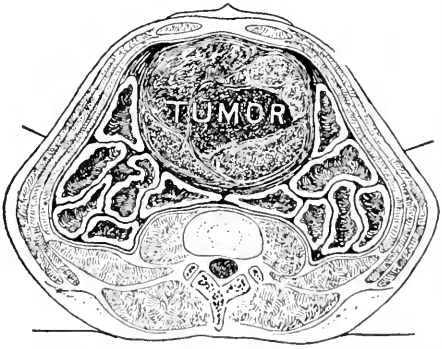


FIG. 78.—Abdominal tumor—increase in anteroposterior diameter—flatness centrally and tympany in flanks.

on the contrary, pushed aside, and causing resonance in the flanks, which does not change to any great extent with change of position and does not present the oval area of tympanitic resonance in the umbilical region which is characteristic of ascites. The presence of peritoneal adhesions and a great quantity of fluid sometimes renders fluctuation obscure and the results of percussion uncertain by interfering with the free movement of the supernatant intestines. Tumors of sufficient size to give rise to marked distention and general dulness or flatness usually increase the anteroposterior diameter of the abdomen to a greater extent than the bilateral as compared with ascites and meteorism. The enlargement caused by tumor is not usually symmetrical. The intestines are pushed aside and tympanitic resonance is

elicited upon percussion in the flanks and especially upon the opposite part of the abdomen to that from which the growth has developed—on the left side in case of tumor of the liver, on the right in case of tumor of the spleen, above in tumors springing from the pelvic organs, below in those springing from organs in the epigastric zone, and so forth, while over the tumor there is dulness. The list of tumors which attain dimensions sufficiently great to cause general distention of the abdomen comprises cancer, syphilitic and amyloid disease, and hydatid cysts of the liver; malignant disease and multiple cysts of the kidney; cancer of the intestines and peritoneum; ovarian cysts and uterine fibromata and retroperitoneal sarcoma. Very marked distention may be present in pancreatic cyst, hydronephrosis and tuberculosis of the mesenteric glands, and Hodgkin's disease. All these conditions yield dulness upon percussion. An important sign in the diagnosis of large neoplasms of the retroperitoneal glands arises from the fact that, while the intestines are in general pushed aside by the tumor, the ascending or descending colon, according to the side upon which the growth develops, passes obliquely across it and yields tympanitic resonance, at both borders of which dulness begins.

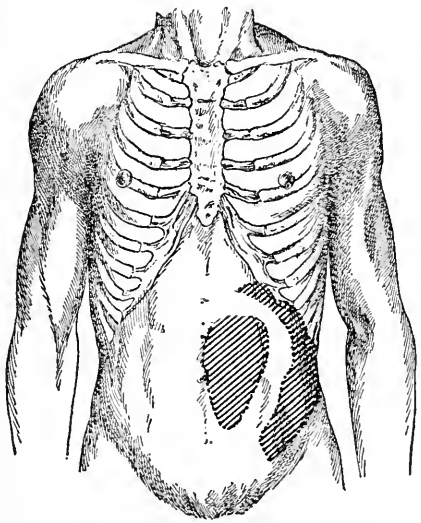


FIG. 79.—Tumor of left side of abdomen—dulness with strip of tympany corresponding to colon.

Gaseous distention of the abdomen—meteorism, tympany—yields tympanitic resonance at all points and increases the vertical diameter of the abdomen by pushing the diaphragm upwards and interfering with its descent. In extreme distention the note becomes higher in pitch, shorter in duration, and diminished in intensity until it finally may be dull. This condition is commonly due to paresis of the intestinal wall and occurs in peritonitis, the advanced stages of the infectious fevers, and hysteria. To a less degree it is present in cretinism, rickets, and pseudohypertrophic paralysis. Great dilatation of the stomach and congenital dilatation of the colon are attended with general abdominal enlargement over which the note is tympanitic.

Free gas in the peritoneal cavity may be the outcome of a perforating ulcer of the stomach or duodenum—peptic ulcer—or of the ileum in enteric fever or of the appendix. The accident which leads to the escape of gas is usually attended with severe abdominal pain, collapse, and meteorism. Rapid obliteration of liver dulness in an abdomen not previously much distended is an important sign. Mere disappearance of the anterior liver dulness at the margin of the ribs or in the nipple line may be a sign of ordinary meteorism. If, however, liver dulness is present in the infra-axillary line while the patient is in the dorsal decubitus and is replaced by tym-

panitic resonance when he is turned upon the left side, it may be inferred that there is free air in the peritoneal cavity.

Local Areas of Dulness.—Spasmodic local contractions of the abdominal muscles and phantom tumors may yield dulness upon percussion. In obscure cases a somewhat deeply seated tumor may be examined by percussion, if the walls are relaxed, by pressing with the pleximeter hand gently but firmly for a time until the bowel is pushed aside, and the mass may be recognized by palpation and its percussion signs ascertained. Any local tumor or new growth gives rise to percussion dulness in that area of the abdominal wall which overlies it. The variety of such morbid conditions is very great. The nature and point of origin of the most important of them have been indicated under the heading Palpation in Diseases of the Abdominal Organs.

AUSCULTATION.

Auscultation as a method of physical diagnosis is the art by which we recognize and interpret the sounds produced within the body in health and disease.

This is the most important of the methods of physical diagnosis. It is essential to the diagnosis of diseases of the organs of respiration and circulation and of limited service in the diagnosis of diseases of the digestive organs.

The Methods.—Auscultation is of two kinds,—*immediate or direct*, in which the ear is applied directly to the surface to be examined, and *mediate or indirect*, in which a stethoscope is employed. The latter was practised by Laennec, the discoverer of auscultation; the former has since come into use.

Each of these methods has its peculiar advantages in diagnosis. Direct auscultation is useful for a general survey of the chest, including both its respiratory and circulatory phenomena, the study of broad areas and the determination of the presence or absence of abnormal signs. It also enables us to detect the signs of deep-seated lesions, as central consolidation of the lung, which are not audible by the stethoscope. Indirect auscultation, on the other hand, is preferable for the nice study of the signs heard in limited areas, the point of maximum intensity of a murmur or the limits of a friction sound. Just as in palpation we use the palm of the hand to find and estimate the extent of the impulse of the heart and then study its force and characters with the smaller and more sensitive finger-tips, so the experienced diagnostician uses the two methods of auscultation. Like the other methods of physical diagnosis they are not independent and sufficient of themselves, but interdependent and complementary. There is no question as to which should be employed, since both are necessary: the one for one kind of observation, the other for a different kind; the one for clinical research, the other to control its results.

Many experienced auscultators use the direct method in the examination of the back of the chest and the stethoscope for the examination of the anterior surface, the reason for this being found in the difficulty in reaching the supraclavicular and axillary regions by the direct method, the closer study necessary in the examination of cardiac and pericardial

conditions, and certain personal considerations which appeal to the user of the stethoscope.

Stethoscopes.—These instruments are made of various materials and shapes. The young auscultator of a mechanical turn of mind is very apt to turn his attention to the stethoscope and there are many inventions. Few only deserve serious consideration. The underlying principle is the conduction of the sound. There are two kinds of stethoscopes, the single and the double or binaural.

The single stethoscope was used by Laennec. The best form is the gun-metal instrument with detachable hard-rubber ear-piece devised by Hawksley of London.

The double stethoscope of Cammann of New York consisted of a chest-piece connected with two tubes fitted with ear-pieces. Many modifications of this instrument have since been made and the double stethoscope has come into general use. The chest-pieces as now made consist of interchangeable bell-like expansions of hard or soft rubber, or a shallow metal cup with a hard-rubber diaphragm held in place by a metal ring, seven-eighths of an inch in diameter so as to be applied to the costal interspaces, or larger; the tubes are long and flexible to enable the examiner to move the chest-pieces freely without changing his position, while the ear-pieces are in some instances attached to metal arms held together by a spring or hinged and held in position by a rubber band. In other forms the soft-rubber tubes are connected directly with the chest-piece and ear-piece, the latter retaining its place in the meatus by its appropriate shape and size.

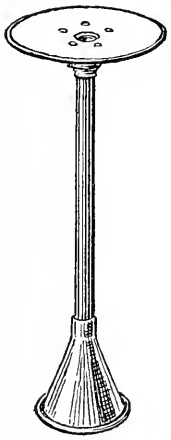


Fig. 80.—Hawksley's single stethoscope.

In selecting a stethoscope attention should be given to the kind. It should be an excellent conductor of sound as tested by comparing several different instruments under similar conditions, and simple in construction, durable, and convenient to carry. Attention must also be given to the particular instrument to see that the ear-pieces fit comfortably, that the pressure is right, and that extraneous sounds are excluded.

With a good instrument, even with the unaided ear, and a fair amount of training the sounds which constitute auscultatory signs may be heard. The problem in diagnosis is their proper interpretation.

The phonendoscope of Bianchi consists of a shallow metallic circular chest-piece with vibrating hard-rubber disks and soft-rubber tubing conductors to the ear-pieces. It is readily applied, and, while it intensifies the sounds, does not produce exaggerated sounds. It is especially useful in auscultatory percussion.

In the Bowles stethoscope the chest-piece is constructed with a vibrating hard-rubber diaphragm with the attachment for the conducting tubes at a right angle to its central axis. Multiple attachments are made for class demonstration. The sounds are intensified and the claim has been made that cardiac murmurs otherwise inaudible may be distinctly heard. The flat chest-piece is especially serviceable in the examination

of the back of the chest in persons too ill to be moved, since it may, by pushing the bedclothes down, be slipped under the patient's back at different points without changing his position. Combination forms in which the Bowles attachment is fitted into the chest-piece of an ordinary stethoscope are sold.

The differential stethoscope of Alison has two chest-pieces with separate conducting tubes. This instrument enables the examiner to compare the sounds heard at different parts of the chest and to study differences in their acoustic properties as well as in the time of their occurrence. Notwithstanding its value it has not come into general use.

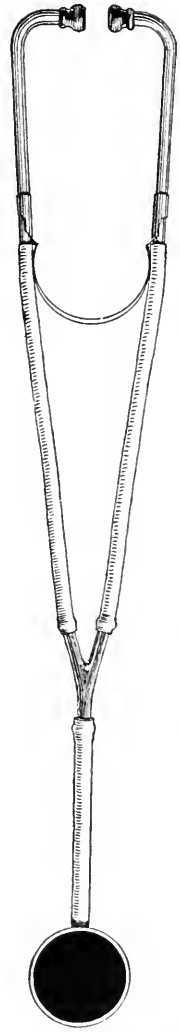


FIG. 81.—Bowles binaural stethoscope.

The Technic of Auscultation. — The following general rules are to be observed:

1. The patient's attitude should in so far as possible be comfortable and unconstrained.

2. Let the chest be bared or covered only with a towel or single layer of undergarment. When the stethoscope is used it is better to have the chest bare. When direct auscultation is practiced it is convenient and fitting, though not essential, to have a layer of thin stuff between the ear of the examiner and the skin of the patient. Auscultation cannot be properly performed through the patient's ordinary clothing. The superimposed layers of several garments, silk fabrics, and the suspenders or corsets not only mask the sounds within the chest but also give forth sounds of their own upon respiratory movements.

3. In indirect auscultation apply the chest-piece of the stethoscope closely to the surface, steadying it by grasping it between the thumb and index finger.

4. If the single stethoscope is used, it must be applied perpendicularly to the surface. If it is tilted, external sounds are not excluded.

5. The stethoscope must be applied very lightly in auscultation of blood-vessels. The rim of the instrument may cause a murmur in the vessels at the root of the neck or in the abdominal aorta by causing the physical condition to which such murmurs are due, namely, sudden narrowing of the lumen—stenosis.

6. Examine the chest in a routine manner first at one apex, then at the other, and at corresponding points upon the two sides from above downwards, in front, behind, and at the sides. Comparison and contrast are essential to auscultation. Equally important are the differences in the sounds upon ordinary quiet breathing, full respiration, and coughing. The respiratory signs are to be considered also in connection with the signs upon auscultation of the voice. In very serious cases, where the patient cannot be disturbed or where the condition can be at once recognized, a complete systematic examination may be omitted.

7. Examine the heart in the same systematic manner, placing the stethoscope over the puncta maxima in turn and noting the direction in which sounds or murmurs are propagated together with the presence or absence of friction sounds, etc.

8. Consider the patient. Do not fatigue him unnecessarily either in mind or body. Do not cause distress by undue pressure of the stethoscope or by insisting upon the repetition of deep breathing or cough when they give rise to pain. Conduct the examination with method, dispatch, and regard for his feelings and do not repeat it with unnecessary frequency.

9. Consider yourself. Assume a position which enables you to place your ear or the stethoscope in accurate relation to the surface to be examined. Use such patience with skill as will render the examination satisfactory to you. If, despite your efforts, the results do not justify a diagnosis, defer expressing an opinion until you have an opportunity of repeating the examination under more favorable circumstances. In dispensary and hospital practice be on your guard against vermin.

In children auscultation is even more valuable in the diagnosis of diseases of the chest than in adults. Owing to the great elasticity of the walls of the chest and the corresponding increase of resonance, percussion is of much less general applicability. Dulness, even when the physical conditions which cause it are present, is not usually so marked nor its limits so easily recognized, nor do we derive the same advantage from comparing and contrasting the two sides, since the acute pulmonary affections of early life are much more frequently double than those after the second dentition.

In children the back of the lungs should be first listened to. The diagnosis may often be made at once upon a careful and systematic examination of the back alone, after taking the history of the illness and noting the symptoms. This is especially true in acute and chronic bronchitis, croupous and bronchopnéumonia, and pleural effusion. Crying is of great assistance. The deep inspirations develop the signs characteristic of the lesions which are present, and we also obtain the signs which arise from the character and modifications of the vocal resonance.

The position in which the child is examined by auscultation should vary with its age. Very young infants may be examined in either a lying or sitting posture on the lap of the nurse or upon a pillow; or they may be held in the arms of an attendant who presents one part of the chest after another to the ear of the physician. The physician himself may hold the baby seated upon his left hand and supported by his right hand applied to the front of its chest and listen to its back with his right ear. Older children may be held seated upon the forearm of the mother or nurse with the head resting upon her shoulder while the physician listens to the back.

The difficulty with beginners in auscultation is that they hear too much. They cannot at first discriminate between sounds that are significant and those which are irrelevant. The power to do this comes, however, with practice.

The most important of the sounds which, by a process of selective attention, the young auscultator must learn to disregard are the following:

1. *Outside Noises.*—A quiet room and silence are desirable but cannot always be secured. We must train ourselves not to hear extraneous sounds while engaged in listening to those which are the object of our immediate attention. Properly fitting ear-pieces and accurate adjustment of the chest-piece of the stethoscope are of help in excluding the sounds which we do not want to hear.

2. *Accidental noises* produced by the stethoscope. These comprise friction rubs of the instrument upon the skin, especially when it is dry and harsh or covered with coarse hair; friction rubs of the auscultator's fingers, or his sleeves, or the like, upon the stethoscope; friction or fine snapping sounds caused by the movement of one part upon another of an adjustable stethoscope of several pieces; sounds made by the breath of the examiner upon the rubber cross-piece or steel spring of the stethoscope, and finally the humming or buzzing sound—tinnitus—made by the ear-pieces. Most of these sounds are easily recognized and avoided. The last is to be diminished by very careful adjustment of the ear-pieces and overcome by usage.

3. *Adventitious sounds* conducted by the stethoscope but not properly constituting auscultatory signs. The sounds made by the friction of the clothing and coarse hairs are very confusing. The first are easily obviated; the second by practice, by applying the chest-piece beyond the borders of the hairy patch, or by the use of oil. Sounds produced by the contraction of muscular masses may often be heard, especially upon deep breathing, in various parts of the chest and in particular over the pectorals and trapezii. These sounds are faint and variable in kind but often quite distinct. They can be produced upon the forcible contraction of any muscle. The thenar mass, for example, when contracted with the stethoscope applied over it, affords a good illustration of such sounds. Cabot has suggested that auscultatory sounds described as "crumpling," "obscure," "distant," and "indeterminate" râles are in reality due to muscular contractions. The fact that such sounds are very often associated with distinct or easily recognized râles and other evidences of pulmonary disease and occur in individuals with atrophic chest muscles, should put us on our guard against hasty conclusions.

Auscultation as Applied to the Diagnosis of Diseases of the Organs of Respiration.

It is of practical importance that the movement of the tidal air on quiet breathing is in many persons not sufficient to cause auscultatory phenomena of significance. It becomes necessary then to listen to the chest during deep or forced respiration. If the patient is stupid or awkward, difficulties arise. He holds his breath, or pants, or makes strange noises, or does not appear able to take a deep breath. You show him how to breathe for the examination or you ask him to cough, listening to the respiratory signs during the deep inspiration which follows or precedes, or you ask him to count as long as he can with a single breath. The full breath which follows enables us to ascertain the presence or absence of abnormal signs. These difficulties are usually encountered in subacute and doubtful cases. In acute cases and in chronic cases with advanced lesions the signs are commonly distinctive upon ordinary breathing.

THE SIGNS IN HEALTH.

Auscultation of the normal chest discovers two respiratory sounds which are typical:

1. *Tracheal, bronchial, or tubular breathing,*

2. *Vesicular breathing,* and combinations of these types in varying degree, namely,

3. *Bronchovesicular breathing.*

1. **Tracheal, bronchial, or tubular breathing** is heard when the stethoscope is placed over the thyroid cartilage, over the trachea in the episternal notch, and in the upper part of the interscapular space upon the right side—normal bronchial respiration. Sometimes nearly pure bronchial breathing can be heard in health over the manubrium sterni or the three lower cervical vertebræ.

It has its origin in the larynx, and is sometimes for that reason spoken of as laryngeal, and, from the situations at which it is heard in health, tracheal or bronchial. Since it is conducted along the column of air in the bronchial system and probably also along its elastic walls and resembles the sound produced by breathing through a tube, it is called tubular.

This type of breath sound is heard with inspiration and expiration, these two elements of the sound being separated by a brief interval of silence at the end of inspiration. Its quality is bronchial, tubular, or blowing; its pitch relatively high as compared with vesicular breathing. The expiratory element is slightly more intense, usually of higher pitch, and slightly more prolonged than the inspiratory part. It may be imitated by slowly breathing through the hollow of the hand, closed by flexing the fingers till their tips touch thenar and hypothenar eminences, or through the lips and teeth held in the position to sound the German *ch*.

It is produced at the chink of the glottis where the air upon inspiration and expiration is thrown into eddies or swirls—*fluid veins.* For the reason that a similar mechanism is involved in the production of vesicular breathing and cardiac and vascular murmurs, it may properly be considered at this point.

The Theory of Fluid Veins. — Chauveau pointed out the fact that when a fluid is forced under pressure from a narrow into a wider tube or channel, or through a narrow opening into a large cavity or space, it is thrown into swirls or eddies, the vibrations of which, transmitted to the enclosing substance and to the surrounding air, are recognized as auditory phenomena. These swirls have been called fluid veins. They are currents within currents, and their vibrations are not only transmitted laterally but also longitudinally in the stream in which they exist, so that the sounds are heard over the point at which they are produced and at a distance in the direction of the flow. The extent and force of these swirls and the consequent loudness of the sound by which they are represented depend to some extent upon the composition and density of the fluid but mainly upon the force of the current. The student will realize the nature of fluid veins and the part they play in the production of the bronchial respiration and the vesicular murmur,—for the air acts in the same way as other fluids,—and especially their part in the production of endocardial and vas-

cular murmurs, if he considers the course of a rivulet which flows at one time down a steep and rapid course, and at another along a nearly level bed with even sides and a smooth bottom, and now as a gentle stream and again with considerable force. The quiet current flowing in even banks is smooth and noiseless, while the little torrent in its rocky bed has its surface thrown into countless screw-like swirls, and murmurs or roars, according to the force and volume of the water. The stream is an open channel; the respiratory and vascular spaces are closed tubes; but the mechanism by which the sounds are produced is the same in each. It is evident that the intensity of the bronchial respiration will vary with the quantity of the tidal air, the force with which it passes through the glottis, the distance at which it is heard, and the conducting properties of the media through which it is transmitted. Variations in pitch depend upon the size and shape of the spaces—pharynx, buccal cavity, trachea, etc.—which constitute resonating chambers in relation with the larynx. We are thus prepared to find wide differences in intensity and pitch in the breath sounds which have the characteristic tubular or bronchial quality.

2. **Vesicular Breathing.**—Respiration of this type is heard when the stethoscope is placed elsewhere over the chest where the lungs are in contact with the chest wall, namely, in the front of the thorax with the exception of the area of superficial cardiac and hepatic dulness, in the infrascapular regions and in the axillary and the upper part of the infra-axillary regions. In the right interscapular region the breathing in health is usually bronchovesicular, the vesicular element predominating.

This sound has its origin in the parenchyma of the lung, and is due to the transmission of the vibrations, caused by fluid veins or swirls in the air passing into and out of the infundibula and alveoli, to the surface of the chest. The hypothesis that the vesicular respiration is merely a modification of the bronchial appears to me to rest upon insufficient facts. This type of breathing is heard throughout the whole act of inspiration, and is immediately followed, without an interval of silence, by a short but inconstant expiratory sound. The inspiratory portion is low in pitch as compared with bronchial respiration, of variable intensity, and has the characteristic quality described as vesicular, which is to be learned only by experience. It is sometimes called the vesicular murmur, and it may be of service to the student to note that it possesses the distinguishing peculiarity of murmurs, namely, that they are sounds made up of a multitude of small sounds, all having about the same acoustic properties, as we speak of the murmur of a crowd, of the leaves of the forest, of the sea, and so on. The expiratory part is still lower in pitch than the inspiratory, much less intense, frequently absent altogether, and does not exceed one-third the length of the latter.

The vesicular murmur is not equally intense in all parts of the chest. It is loudest in the infraclavicular, axillary, and infrascapular regions, and fainter at the bases in front and behind. That is to say, it is loudest over large masses of lung tissue and faintest over the thin wedge-shaped borders. But it is also less distinctly heard in the mammary and scapular regions. We conclude therefore that it is not well conducted through thick layers of muscle, bone, and fat. Wherever heard, whether loud or faint, it retains

its characteristic breezy quality and low pitch, and the relative duration, intensity, and pitch of the inspiratory and expiratory elements are preserved.

The vesicular murmur is feeble and distant on shallow breathing and intense upon deep breathing, especially after prolonged deep breathing as after exertion. It is intense over the unaffected lung in cases in which the opposite lung has been thrown out of service by disease, and in healthy children, hence it is spoken of, when thus intensified, as "puerile" or "exaggerated" respiration. Intense vesicular respiration somewhat modified is spoken of as "rough"; just as bronchovesicular respiration is often called "harsh."

3. **Bronchovesicular Breathing.** — This form of respiration, as the name indicates, has the characteristics of both bronchial and vesicular breathing and consists in fact of a breath sound in which both are present. It is heard in the normal chest very often, but not invariably directly below the right clavicle, and quite constantly at the sternal borders opposite the lower part of the manubrium and in the upper portions of the interscapular spaces, namely, in situations in which both sounds are within range of hearing. Many of the difficulties regarding bronchovesicular respiration are solved when we recognize the fact that it is made up of the two forms in varying degrees of combination, so that it sometimes presents the traits of bronchial breathing slightly modified by the admixture of faint vesicular breathing and sometimes those of vesicular breathing slightly modified by bronchial, and between these two we encounter every grade of admixture. This gradation by which the breath sound passes from bronchial to the vesicular respiration may be heard in the normal chest by moving the stethoscope from point to point, starting at that part of the manubrium over which bronchial breathing is heard and advancing towards the nipple where the vesicular murmur alone can be recognized. The inspiration becomes, as we proceed, lower in pitch, less intense, and longer in duration, and the expiration also lower in pitch and less intense, but shorter in duration. The interval of silence which is characteristic of bronchial respiration is filled by the vesicular element in bronchovesicular respiration. This interval of silence is present in bronchial breathing because the swirls —fluid veins—by which the vibrations causing the sound are produced, arise at a single point, the glottis, and there is at that point an interval of equilibrium between the flood tide of inspiratory and the ebb tide of expiratory air. The vesicular murmur, on the other hand, is produced at a multitude of different points, and the moment of silence is as variable as the individual little sounds which cover the whole time of the inspiratory act, since vesicles at the distant periphery of the lung are still expanding when those nearer the inlet have ceased to dilate.

The conditions which modify the bronchial respiration as a physical sign and those which modify the vesicular murmur also modify the bronchovesicular breathing. It therefore presents differences in intensity, duration, and pitch, corresponding to variations in the quantity and force of movement of the tidal air, to the size and shape of the resonating chambers formed by the upper air spaces and the tracheobronchial system and the physical condition of the intervening tissues through which the sounds are conducted to the ear. The qualities of the two component types of breathing, though they vary in proportion, are not changed.

It is essential for the student to become familiar with these three forms of normal breath sounds and the localities in which they may be heard in the normal chest. Familiarity with normal physical signs is the first step towards the recognition of those which are abnormal.

Bronchial breathing is heard in the front of the neck and over the upper part of the manubrium, vesicular breathing over the greater part of the chest, as above, because the mechanism by which they are respectively produced is situated in the regions indicated. Bronchovesicular respiration is heard normally over the lower part of the manubrium and laterally to it and in the interscapular spaces because both its factors are within the range of hearing. Bronchial respiration is heard in the right interscapular space and bronchovesicular respiration is more prominent (bronchial) over the upper part of the right lung by reason of the larger size and higher origin of the large bronchus on the sight side.

Bronchial or tubular breathing is conducted in the column of air in the bronchial tree to its remote twigs. It is not conducted to the surface of the chest because the vibrations are on the one hand lost in the mass of cushiony, elastic vesicular tissue which constitutes the lung parenchyma, and on the other hand the bronchial sound is drowned in the vesicular murmur. When this tissue becomes solidified by compression—atelectasis—or by an exudate—pneumonia, tuberculosis—the vesicular murmur is done away with and the vibrations conducted by the bronchial tubes are freely transmitted to the surface.

THE SIGNS IN DISEASE.

The auscultatory phenomena which constitute abnormal or morbid physical signs are (a) variations in the intensity and rhythm of the breath sounds, (b) normal physical signs heard in abnormal situations, and (c) purely adventitious sounds.

(a) **Variations in Intensity and Rhythm.—Bronchial Respiration.**—It has been explained that bronchial respiration heard beyond the limits of certain regions of the chest in which it is normally present is usually due to the consolidation of lung tissue—atelectasis; presence of an exudate, as in pneumonia, tuberculosis, etc. It may, however, arise in connection with cavities in the lungs or pneumothorax. Under these circumstances there are layers and masses of compressed or consolidated lung tissue present and the peculiar modification of the bronchial respiration is probably due to the fact that the cavity acts as a resonating space. Bronchial respiration varies greatly in pitch. This variation is the outcome of complex conditions not fully understood, but has been attributed to the relative size of the tubes or cavities from which the sound is directly conducted through consolidated tissue to the ear. The pitch is usually high and the sounds whiffing or snoring in pneumonia of the lower lobes, especially in children, and low and the sound soft and sighing or metallic over cavities.

The following varieties of bronchial respiration are to be especially considered:

1. *Feeble and distant bronchial respiration* is often heard in central pneumonia and pulmonary infarct and over a pleural effusion. In the

former case the bronchial breathing may be only heard upon deep inspiration and is therefore inconstant; in the latter it is frequently so faint as to be overlooked. The sound is conducted by the chest wall or by tense adhesions, the result of former attacks of pleurisy.

2. *Intense bronchial breathing* usually conveys the sensation of being close to the ear, that is, well conducted. It accompanies dense consolidation of the lung in which vicarious or supplemental respiration is well established.

3. *Absence of bronchial respiration* or its sudden disappearance under conditions in which the mechanism for its conduction exists may be due to the plugging of a large bronchus with a mass of tenacious exudate. The disappearance of cavernous or amphoric respiration often results from the accumulation of fluid within the walls of the cavity. Under these circumstances the bronchial respiration returns after cough and expectoration.

4. *Cavernous respiration* is a variety of bronchial breathing sometimes heard over a cavity. It is low in pitch, soft in quality, and the expiratory element is prolonged.

5. *Amphoric respiration* is a variety which has the peculiar quality heard when one produces a sound by blowing across the mouth of an empty jar or bottle. The pitch is variable, usually low, and the sound is hollow, metallic, and musical. Amphoric respiration is never heard over the normal chest, and indicates a superficial cavity with rigid walls—or pneumothorax —having free communication with a large bronchus. The sound may be imitated by whispering "who" with some force and the lips held rigid.

Vesicular Respiration — The normal vesicular murmur undergoes modifications in intensity and rhythm which are of diagnostic significance.

1. *Feeble vesicular respiration* primarily indicates diminution in the quantity and energy of the movement of the tidal air. Hence it is present in varying degrees in quiet breathing in aged and bed-ridden persons, in paretic conditions of the respiratory muscles, including the diaphragm, when the movement of the diaphragm is impeded by meteorism, ascites, abdominal tumor, or pregnancy. The vesicular murmur is often feebly heard because it is poorly conducted, as in very thick chest walls. In pleural adhesions the expansion of the periphery of the lung may be embarrassed, and with thickening conduction is also impaired. A thin layer of effusion or a tumor acts in the same way. In pneumothorax the lung is compressed and removed from contact with the chest wall, and the vesicular murmur, if heard at all, is faint and distant. In acute bronchitis the swelling of the mucosa and the presence of the exudate interfere with the access of air to the vesicles and proportionately enfeeble the vesicular murmur, especially over the lower lobes. In chronic bronchitis enfeeblement is brought about by the accompanying emphysema and restricted movements of the chest.

In congestion and œdema of the lungs the murmur is enfeebled.

Emphysema by impairing the elasticity of the lungs and restricting the respiratory excursus increases the residual and diminishes the tidal air, thus rendering the vesicular murmur faint and in rare cases almost wholly abolishing it. Pain, as in pleurisy, restricts the respiratory movement and renders the vesicular sound faint.

Occlusion of the upper air-passages, as by spasm, œdema of the glottis, the presence of an exudate, as in diphtheria, quinsy, or retropharyngeal abscess, renders the murmur feeble in proportion to the extent of the obstruction. Pressure upon the trachea or a primary bronchus by aneurism, tumor, or enlarged lymph-gland acts in the same way. A foreign body or a plug of tenacious mucus in a bronchus enfeebles the respiratory murmur in the corresponding region to a degree proportionate to the stenosis. In such conditions the occurrence of râles obscures the enfeeblement of the respiratory sounds and the latter will be overlooked unless made the subject of especial attention.

2. *Absence of the vesicular murmur* may be noted over an area of the chest more or less extensive when the obstruction to a bronchus in any of the foregoing conditions is complete. Marked obstruction of the upper air-passages is at once followed by the signs of asphyxia. The murmur is absent over the greater part of the chest in rare cases of advanced emphysema, and no respiratory sound is heard over a pneumothorax not communicating with a bronchus, a large pleural effusion and locally over limited areas in some cases of cirrhosis of the lung and at the apex in rare instances in beginning tuberculosis.

3. *Intensified or exaggerated vesicular breathing—puerile, vicarious, or compensatory respiration*—is normal in childhood and gradually decreases until some time before puberty the intensity of the sound becomes that of adult life. It occurs in health after exertion and in dyspnœa from almost any cause in which there is no obstruction to the entrance of air. It occurs over one lung when the other is put out of service, as in pneumonia, large effusion, tumor, etc., and in some instances over a portion of one lung under similar conditions, hence the adjectives vicarious and compensatory.

4. *Derangements of rhythm occur in emphysema*, in which the loss of elasticity relatively prolongs the expiratory act and the expiratory sound; in asthma, in which the dyspnœa is expiratory, in the ordinary dyspnœa or panting of great exertion, in which the inspiratory and the expiratory breath sounds are nearly equal, and in various forms of inspiratory dyspnœa which are attended by diminution of the intensity and prolongation of the inspiratory element of the vesicular murmur.

5. *Interrupted or cogwheel inspiration* is characterized by a series of two, three, or four inspiratory sounds instead of the normal continuous murmur. It indicates in some instances a fault in the muscular function and occurs during periods of excitement or during a chill; more commonly it is a sign of early pulmonary tuberculosis, the air entering adjacent lobules in turn as the force of inspiration increases. It is usually heard in limited areas. When restricted to the precordial space it is significant of pressure of the heart upon the borders of the lung—cardiopulmonary murmur. In some instances the respiratory sound is not actually broken, but wavy or jerky, and is then described under these terms. It is not rarely present in tuberculosis before the disease has shown itself by other signs, and individuals who present it should be carefully watched. In other cases it is wholly without diagnostic significance, which it acquires only in conjunction with other physical signs or the symptoms of pulmonary disease.

(b) **Normal Physical Signs in Abnormal Situations.—Normal Sounds Heard in Abnormal Situations.**—Note the relative duration of the inspiratory and expiratory sounds and determine the presence or absence of an interval of silence between them and the quality of the sound, whether soft and breezy—vesicular murmur; blowing and tubular—bronchial breathing; or whether these qualities are both present—bronchovesicular. The most important facts for the beginner in the recognition of bronchovesicular respiration are the prolongation and relatively high pitch of the expiratory sound.

Perfectly normal vesicular respiration is rarely heard in other than its extensive normal domain in the chest. The rare cases of dextrocardia are attended with dislocation of the precordial space, and fibroid contraction of one lung frequently displaces the border of the opposite lung towards the affected side so that it occupies the area of superficial cardiac dulness in whole or in part. The modified respiration of emphysema, faint and prolonged, is sometimes heard in the precordia and over the upper normal area of the liver dulness.

Bronchovesicular and bronchial respiration are on the contrary common and significant signs of disease in the chest. The lesions are commonly progressive, and bronchovesicular usually, both in acute and chronic affections, precedes and progressively develops into bronchial respiration. Pulmonary consolidation either from compression or infiltration is the underlying physical condition and reaches its extreme development whether rapidly or slowly by progressive advance.

These signs are heard over the compressed lung in the following conditions: pleural effusion, the area in which they are present becoming more limited and the respiration more characteristically bronchial as the effusion augments; pericardial effusion; pneumothorax, in which more or less complete compression of the lung, unless prevented by old partial adhesions, takes place rapidly; tumor of the lung or pleura; massive enlargement of the heart, and large aortic aneurism. They are heard over the lung undergoing solidification or already solidified from infiltration in tuberculosis, bronchopneumonia, croupous pneumonia, pulmonary infarct. As already pointed out, distant bronchial breathing may frequently be heard over an effusion. It remains to point out the more important fact that loud, distinct, and well-conducted bronchial respiration is by no means uncommon over pleural effusions of large amount in thin-walled individuals and especially in children. This sign is conducted from the compressed lung by way of the wall of the chest and probably in some cases also along bands of old adhesions tightly stretched between the compressed lung and the chest wall by the force of the accumulating fluid. In pneumothorax the variety of bronchial breathing known as amphoric is heard when there is free communication between the pleural cavity and a bronchus.

Cavernous or amphoric respiration may be heard over cavities, whether due to the breaking down of lung tissue (tuberculosis, abscess, gangrene) or to dilatation of bronchi (bronchiectasis). Deep-seated cavities due to any of these causes may be attended with distinct bronchial respiration yet be difficult to locate with precision.

Bronchovesicular respiration must be distinguished on the one hand from puerile or exaggerated vesicular respiration and on the other from

bronchial respiration. The breezy quality, low pitch, short expiratory element, and absence of a period of silence between inspiration and expiration are characteristic of the former, however intense. The tubular quality, relatively high pitch, longer expiratory sound, and an interval of silence are distinctive of the latter, and between the two are all degrees of transitional sounds. Normal in the right infraclavicular region, at the upper sternal borders, and in the neighborhood of the upper dorsal vertebræ, bronchovesicular respiration elsewhere in the chest becomes a sign of disease, and denotes partial consolidation of the lung, patches of collapse or infiltrated lung or consolidation with intervening normal vesicular tissue. This sign is present in early pulmonary tuberculosis, at the borders of the exudate in croupous pneumonia, in bronchopneumonia, and in incomplete atelectasis from any cause.

(c) **Adventitious Sounds.**—Purely adventitious respiratory signs are of two kinds: (1) Râles, which are produced by abnormal conditions within the lungs, and (2) friction sounds which originate in the pleura.

1. **Râles.**—Literally a râle is a "rattle" and may be defined as an abnormal respiratory sound heard on auscultation. Râles are grouped as dry or moist according to the impression conveyed to the mind as to the presence or absence of fluid in the mechanism by which they are produced. They are laryngeal, tracheal, bronchial, vesicular, and cavernous, according to the situations in which they occur.

In general râles or rhonchi are generated in the air-passages by the ebb and flow of the air when their lumen is contracted or when they contain fluid—dry and moist bronchial râles. Certain râles originate in the bronchioles and vesicular structure of the lung (vesicular râles), others in cavities (gurgling), and finally the succussion sound and the sign known as *gutta cadens* or *metallic tinkling* have their origin in hydropneumothorax. Râles may be heard upon inspiration or expiration or during both acts. They may obscure the normal breath sounds or entirely replace them.

Dry râles are produced by stenosis of the bronchial tubes. This narrowing may be present at one point only as in laryngeal diphtheria or œdema of the glottis, or a tumor pressing upon the trachea, but is usually present at the same time at many points and in many bronchial tubes of varying diameter. It is brought about by a variety of pathological conditions, as a mass of tenacious mucus adherent to the surface of the tube, swelling of the mucosa or submucosa, spasmodic contraction of the bronchial musculature, and the external pressure of enlarged glands or a tumor. When this narrowing involves the smaller bronchial tubes the râles which result are high pitched—*sibilant;* when it affects the larger tubes the râles are low pitched—*sonorous.* They resemble the cooing of doves, the hissing of geese, and have very often a musical quality. Sometimes they are groaning or squeaking. In asthma they are often heard in great variety of size, pitch, intensity, and quality, both upon inspiration and expiration, and appear and disappear with the most remarkable modifications and great rapidity.

Moist râles are caused by the passage of air through the bronchi when they contain fluid—mucus, pus, blood. The mechanism consists in the presence of bubbles or diaphragms before the incoming and outgoing air

which continuously burst and reform. When this process takes place in the larger tubes, the bubbles are large and the *râles coarse or large bubbling;* when in the smaller tubes, they are finer, *small bubbling or subcrepitant râles.* Large moist râles are usually low in pitch; small moist râles higher, and in this respect moist and dry râles correspond. The tracheal râle or *death-rattle* is an example of a very coarse râle; the small moist or subcrepitant râle heard in bronchopneumonia in both respiratory acts is an example of a fine moist râle.

Both dry and moist râles vary in intensity and locality. The extent of the area over which they are heard depends upon that of the process by which they are caused; their acoustic characters upon the physical changes produced by that process. In bronchitis râles are very often best heard at the bases of the lungs posteriorly; in tuberculous disease of an apex, in the subclavicular region. Râles are very often influenced by the act of coughing and expectoration. Dry râles produced by pressure stenosis, tenacious exudate which cannot be dislodged, or bronchial spasm, do not disappear upon coughing.

Vesicular or crepitant râles originate in the finest bronchioles and air-cells. Notwithstanding the differences of view in regard to the mechanism by which they are produced, the weight of evidence is still in favor of the theory that it is by the inspiratory separation of the walls of terminal structures—bronchioles, alevoli—previously collapsed or held together by a thin layer of sticky exudate or serum. In support of this theory the following facts may be adduced: This râle, at one time held to be pathognomonic of croupous pneumonia, is now known to occur also in other pathological conditions in which an exudate or blood is present in the lung parenchyma, as pulmonary œdema, hemorrhagic infarct, and acute pneumonic phthisis. It is common in partial atelectasis—atelectatic crepitation of Abrams. Crepitant râles sometimes associated with subcrepitant râles are frequently heard during deep inspiration at the bases of the chest posteriorly and laterally in persons whose respiration is habitually shallow. This is not only the case in bed-ridden individuals but also in many healthy persons, especially after middle age. The crepitant râle is heard only upon inspiration. The subcrepitant râle with which it is often associated is usually coarser and slightly moist.

The crepitant râle is usually heard towards the end of inspiration; the individual râles are of the same size and intensity and they often occur in "showers," a large number of single sounds having the same acoustic properties following each other in rapid and irregular succession.

The crepitant râle occurs in croupous pneumonia at the beginning of the process,—*crepitus indux,*—disappears when the exudate undergoes coagulation, and reappears together with subcrepitant râles when the exudate undergoes liquefaction and resorption,—*crepitus redux.* This auscultatory sign may be imitated by placing a little mucilage between the finger and thumb and making repeated contact and separation. With contact there is no sound, but upon separating the thumb and finger a string of tenacious mucilage is drawn out which finally snaps with a sharp sound not unlike the râle. It may also be imitated by the crackling of fine salt thrown upon the fire, the creaking of a silk garment, or lightly

rubbing a few strands of hair between the thumb and finger. If the stethoscope is applied over the thick growth of coarse hair found upon the chest of many men, a sound closely resembling crepitation will be heard. Crackling is the term used technically to designate a râle coarser than crepitus but having in other respects similar acoustic properties. This râle consists of a limited number of well-defined sharp crackling sounds often heard in beginning pulmonary tuberculosis or at the borders of an advancing tuberculous lesion and for this reason is of considerable diagnostic importance. The distinction between crepitus and crackling is not always unattended with difficulty. Crepitus consists of a number of fine sounds, heard only upon inspiration and often over a considerable area at the base of the lung; crackling of a few sharp, well-defined, rather coarser sounds heard also in inspiration but over a limited area and commonly at the apex. It is probable that the mechanism is the same in both, but that crackling occurs in limited lesions, hence only a few individual sounds are heard; in wider spaces, terminal bronchi, hence the sounds are coarser; and at a point surrounded by densely consolidated tissue, hence they are better conducted to the ear—consonating râles. Moist crackling and clicking are varieties of crackling which are regarded as indicative of softening tubercle. In certain cases of dry or plastic pleurisy fine dry friction sounds are to be heard which can scarcely be distinguished from subcrepitant râles. If they occur only upon inspiration they may be mistaken for crepitus.

Gurgling or the râle of cavities is caused by the entrance and exit of air in a cavity containing fluid. Coarse churning sounds are heard resembling those produced by pouring fluid rapidly from a bottle. These very coarse, well-defined râles are known also as cavernous, and sometimes have the metallic or amphoric quality.

Metallic Tinkling—Gutta Cadens.—All râles heard in pneumothorax acquire the amphoric or metallic quality. In some instances single râles having an exquisite metallic or bell-like musical quality may follow deep inspiration or the act of coughing. This sound, which resembles that made by single fine shot dropped into a metal bowl or basin, was at one time thought to be caused by a drop of exudate or pus collecting at the vault of the cavity and falling upon the surface of the fluid collected at its base. It is now known that it may occur in the absence of any such collection of fluid and that it may be due to the bursting of a bubble formed at the pleural orifice of a bronchopulmonary fistula.

Hippocratic Succussion.—This phenomenon, although it is not a râle in the narrow sense, may be best described at this point. It is characteristic of hydro- (pyo-haemo-) pneumothorax and consists of a distinct loud splashing which may be heard and felt when the thorax is suddenly shaken. It is due to the swash of the free fluid against the wall of the chest, just as a similar sound is produced by the sudden movement of a partially filled cask.

The Bronchopulmonary Fistula Râle.—In hydro- or pyopneumothorax, when the accumulating fluid rises above the pleural opening of the fistula there may be sometimes heard in connection with paroxysmal cough bubbling sounds due to inspired air being forced from the lung and up through the fluid. Under such circumstances violent spells of cough are apt to be followed by copious expectoration.

Râles may be conveniently grouped as follows:

- Bronchial Râles
  - Dry or Vibrating
    - Low pitched—Sonorous.
    - High pitched—Sibilant.
  - Moist or Bubbling
    - Large bubbling—Mucous.
    - Small bubbling—Subcrepitant.
- Vesicular Râles
  - Crepitus.
  - Crackling (Clicking).
- The Râle of Cavities
  - Very Coarse Bubbling—Gurgling.
  - Cavernous and Amphoric Rales.
- Râles in Pneumothorax and similar conditions. (Hydro-pyo-hæmo-pneumothorax)
  - Metallic Tinkling—Gutta Cadens.
  - The Bronchopulmonary Fistula Râle.
  - The Hippocratic Succussion.

2. **Friction Sounds.**—The surfaces of the normal pleura, being moist and smooth, glide noiselessly over one another with the movements of respiration. When, however, the serous membrane is roughened by the presence of a fibrinous exudate, as in pleurisy, the movement of the opposed surfaces gives rise to sounds known as "pleural friction sounds" or "friction rubs." As the lesions of pleurisy vary from a mere dryness of the surface in the beginning to every grade of exudate in amount, texture, and arrangement, including the fibrinoserous forms, so the friction sounds present great diversity in their acoustic properties, not only in different cases but also in the same case during its course.

The general and almost constant character of pleural friction is, however, that of the sounds produced by the rubbing together of dry or slightly moistened surfaces, and is properly characterized as grazing, rubbing, creaking, leathery, grating, rasping, and the like. Friction sounds are usually jerky and irregularly interrupted, and change in character not only in the course of time but even in the course of a single respiratory act. They are superficial and give the impression of being produced very near the ear. They vary in intensity from a mere graze, scarcely audible, to a coarse, loud, and prolonged creaking like that of new leather and audible to the patient himself or the bystanders. They are described as fine, medium, or coarse. They are as a rule best heard and often only heard in the infra-axillary or inframammary region where the respiratory excursus is widest and the pleura investing the thin wedge of lung is in contact upon one side with the costal and upon the other with the diaphragmatic pleura. Not being well conducted, they are heard where they are produced, so that in cases of diaphragmatic pleurisy the friction sounds may be heard below the level of the lung, in croupous pneumonia opposite the seat of the exudate, and in the earliest days of phthisis at the apex. They may sometimes be heard over the entire lung from the apex to the base. In children and spare persons the intensity of these sounds may be increased by firm pressure upon the chest, and they are often attended by a palpable sign—*friction fremitus*. They occur most commonly during inspiration and especially toward the end of the act, and are frequently heard also during expiration. Less often they are present during expiration alone.

Friction sounds are sometimes inconstant, ceasing after several deep inspiratory acts and being again heard after a period of quiet breathing. They are not modified, however, to the same extent as râles, nor do they

disappear upon coughing and the expectoration of mucus. Various postural methods of bringing out friction sounds in suspected cases have been described, as raising the arm upon the affected side or having the patient quickly rise from the recumbent to the sitting posture during held expiration and then take a very deep inspiration.

Deep breathing, coughing, pressure upon the affected side, not only increase the intensity of the sounds, but are also attended with pain. In exceptional cases friction sounds are unattended by pain during these acts. When a plastic pleurisy is followed by a serofibrinous exudate the friction sounds disappear, but recur upon the resorption or removal of the fluid. They are usually present upon one side of the chest only, but may sometimes, especially in disseminated tuberculosis, be heard in circumscribed areas on both sides.

Crumpling friction sounds are the signs of acute inflammation of the pleura. When the process subsides the surfaces become fused, the fibrinous exudate organized. The condition is that of adherent pleura and, unless dense and extensive, does not give rise to physical signs. In old pleurisy at the apex and especially when cavities exist, curious, low-pitched, soft, creaking sounds are sometimes heard. This sound resembles that produced by squeezing soft thick paper together in the hand in irregular folds and is described as crumpling. It is present upon inspiration and expiration and is not affected by cough, nor has it the characters by which we recognize râles.

In some cases of pleural effusion a considerable period elapses between the resorption of the fluid and the formation of adhesions. During this time friction sounds may be heard and the patient may experience annoying grating or rubbing sensations, especially upon deep breathing or coughing.

Sounds closely simulating friction sounds may be produced by rubbing the thumb and finger together near the ear or by holding the hollow of the hand over the ear and rubbing or stroking the back of it with the fingers of the other hand. There is a fine friction sound which cannot be distinguished from crepitus. Both occur in showers at the end of inspiration, both are close to the ear and have the same acoustic qualities, both are accompanied by an expiratory element which may be in one case a friction sound and in the other a subcrepitant râle. By the sound itself the differentiation is impossible, but when concomitant phenomena are taken into account we find the friction sound is usually more limited in extent, attended more commonly by expiratory sounds, is less uniform in character, and disappears when the movement of the chest wall is restricted by compression, while crepitus persists. The distinction between fine friction of this form and the crepitant râle or crackling is rather of theoretical than practical importance when we reflect that in pneumonia, when, as is commonly the case, the exudate extends to the periphery of the lung, the pleura overlying it is the seat of an inflammatory exudate, and in tuberculosis of the apex the early lesions which give rise to creaking are accompanied by a circumscribed pleurisy. In point of fact when we hear one of these signs the other usually is also present.

The friction sound which closely resembles crepitus or crackling is very rarely, if ever, heard in simple, uncomplicated pleurisy.

Friction sounds heard over the chest are significant of pleurisy. Those over the precordial space, having the cardiac rhythm, are usually but not invariably signs of pericarditis. The subject of pericardial and pleuropericardial friction will engage our attention in a subsequent section. Friction sounds heard in the epigastric zone constitute in rare instances the signs of a peritonitis. The effusion in hydrothorax is not preceded by a friction sound. Pleurisy is frequently primary; often secondary to intrapulmonary disease, pneumonia, tuberculosis, cirrhosis of the lung, abscess, gangrene, or cancer; and sometimes, especially upon the right side, secondary to subdiaphragmatic disease, as abscess, cancer or hydatids of the liver, or subphrenic abscess. Friction sounds may therefore be significant of any of these affections.

Riesman has described under the term subpleural friction a fine soft rubbing or crepitation which occurs in the absence of pain or the signs of consolidation in miliary tuberculosis. The difficulty in distinguishing fine pleural friction from crepitus has already been discussed.

AUSCULTATION DURING PHONATION.

Auscultation of the Voice in Health and Disease.—The sounds heard upon auscultation of the chest of a person who is speaking when the face of the patient is turned away or the opposite ear of the examiner closed, or when the binaural stethoscope is employed, constitute the set of physical signs comprised under the general term vocal resonance, and have diagnostic value. The ordinary spoken and the whispered voice are studied. Obstacles to the employment of this method of physical diagnosis consist in want of coöperation, as in children and extremely ill persons, in inability to use the voice, as in mutes, those suffering from aphonia from any cause, and in extremely feeble patients and great obesity.

The Technic.—The patient counts in a monotone, turns his face away and counts "one, two, three"; or repeats "twenty-one" or "ninety-nine" in the loud voice or in a stage whisper. The sound is conducted through the bronchi and along their walls in the same manner as in a speaking tube and greatly dispersed and damped in the cushiony vesicular tissue. Changes in the physical condition of the lung parenchyma favor or still further impede the transmission of the voice in such a manner that increase, diminution, or absence of vocal resonance correspond to these changes and thus become signs of disease. The modifications of vocal resonance correspond in general to those of vocal fremitus and have the same significance.

Normal Vocal Resonance.—The voice is heard as a confused inarticulate hum, most distinct in adults possessed of deep voices and tremulous in aged persons. This sound is more intense upon the right than upon the left side and at the apices than at the base. As the stethoscope is carried to a position nearer the main bronchi the resonance becomes louder and more distinct until finally, when it is placed over the bronchi or trachea in the position in which normal bronchial breathing is heard, the audible words may be recognized—bronchophony.

Increased Vocal Resonance.—This sign when heard over the lung—with rare exceptions, presently to be mentioned—denotes an increase in

the power of the lung to conduct sound-producing vibrations,—namely, consolidation. It has, therefore, the same significance as bronchial respiration. Fully developed it constitutes bronchophony, and indicates consolidation of lung tissue in the neighborhood of large or medium-sized bronchial tubes. The following varieties are to be considered:

Pectoriloquy.—Laennec used this term to indicate the complete transmission of articulate words. The voice appears to be directly spoken into the observer's ear. This sign occurs in dense consolidation extending from a large bronchus to the wall of the chest, over a cavity communicating freely with a bronchus of some size, in a pneumothorax communicating with a bronchus, and in some instances over the atelectatic lung overlying a large pleural effusion. When pectoriloquy is very distinct and circumscribed it constitutes the distinct physical sign of a cavity, and, as Da Costa well said, deserves the name of cavernous voice.

Amphoric Vocal Resonance. — Over large cavities and in pneumothorax communicating with a bronchus the voice is peculiarly ringing and metallic. The amphoric character is due to the same physical conditions which we find to underlie the amphoric quality in the breath sounds and râles.

Whispering Pectoriloquy.—The whispered voice is heard as a faint, distant, expiratory whiff or puff over the trachea and primary bronchi in front and behind while elsewhere it is almost or quite inaudible. When the physical conditions which cause bronchophony are present, the whispered voice is curiously near and distinct. Whispering pectoriloquy is an important physical sign, indicating, when distinct and circumscribed, a cavity, and in varying degrees of intensity consolidation of lung tissue. It is of value in the diagnosis of limited areas of consolidation and in determining the boundaries of large ones. The more dense the consolidation the more distinct the whispered voice. Whispering pectoriloquy may be present over the atelectatic lung in pleural effusion and occasionally over the effusion itself. Increased whisper in the intrascapular regions—D'Espine's sign—occurs in tuberculosis of the bronchial glands.

Diminished Vocal Resonance. — This sign indicates impaired conduction in the lung and is present in emphysema and the occlusion of a bronchus. It also denotes the interposition of substances between the lung and the chest wall, which leads to the diffusion and weakening of vibrations passing from one medium to another, and occurs in pleural effusion, pleural thickening, and tumors. The more massive the effusion, the greater the thickening, or the larger the tumor, the more marked the diminution in the transmitted voice resonance. It may be completely absent in closed pneumothorax. Absent vocal resonance is most common in large pleural effusion.

Ægophony.—Literally, the bleating of a goat. A peculiar quavering quality of the voice with a distinctly nasal tone is heard when the patient speaks in a natural voice. This sign is best brought out by using repeated rather than single syllables, as "twenty-one" or "ninety-nine." It may be heard at or just below the upper limit of moderate-sized pleural effusions in the region of the angle of the scapula; less frequently in the front of the chest. It is in rare instances heard over consolidated lung tissue. It is not an important physical sign.

Baccelli's Sign.—Upon direct auscultation in the anterolateral region of the affected side the whispered voice is said to be distinctly transmitted through a serous but not through a purulent effusion, the difference being attributed to variations in the density of serofibrinous and purulent effusions. This sign is not constant, since in large effusions there is commonly absence of vocal resonance in both kinds of fluid.

Auscultation as Applied to the Diagnosis of Diseases of the Circulatory Organs.

The Technic.—This method is of cardinal importance in the examination of the heart. Upon it in most instances the diagnosis depends. Inspection, palpation, and percussion may be used to amplify and control the signs obtained by auscultation, but in a considerable proportion of the cases they contribute no essential facts. Before we apply the stethoscope, we inquire into the history of the case and place the patient as far as possible at his ease. The examination is best conducted when the patient is in a comfortable position, leaning back in a chair or propped up with pillows in bed. We note the facial expression, the appearance of the eyes, the state of the capillary circulation, the presence or absence of dropsical swellings, whether or not there is cough, the character of the respiration and any abnormal impulse or movement that may be present at the root of the neck or in the chest. The signs elicited upon inspection, palpation, and percussion are then ascertained. Finally we employ auscultation.

In women the breast is drawn aside and held by the patient herself or her nurse. In young children inspection and palpation should precede auscultation. Percussion is useless. Very often the auscultatory signs must be caught in the intervals of crying and struggling. Many difficulties may be overcome by tact and gentleness.

The increase in the frequency of the heart's action and the accompanying change in the character of the first sound that occur in nervous persons under examination (*le cœur medical*) must be borne in mind. A few minutes' chat upon indifferent subjects will usually cause the excited action to subside. If on the other hand the action of the heart is weak and the sounds too faint to be well studied, or there is a doubt as to the presence of a murmur, the patient should be asked, unless his general condition forbids, to take a series of very deep breaths, or quickly stoop and rise several times, or take a few brisk turns up and down the room. The increase in the force of the heart's action will often render the sounds distinct and dispel any doubt as to the presence of a murmur. In cases of acute disease or profound general or cardiac asthenia such diagnostic measures are strictly contraindicated.

Faint and distant sounds and obscure murmurs may become more audible if the patient leans slightly forward and to his left, thus bringing the heart under the influence of gravity into closer relation with the wall of the chest. It is important also to request the patient to stop breathing for a moment now and again during the course of the examination, since the breath sounds may mask the normal and abnormal

ASSOCIATION OF THE PHYSICAL SIGNS.

Inspection.	Palpation.	Percussion.	Auscultation.	Physical Condition
Normal signs.....	Normal vocal fremitus..	Clear.....	Vesicular murmur or its modifications; normal vocal resonance	Lung tissue normal or nearly so.
Negative.....	Vocal fremitus increased	Vesiculotympanitic.....	Bronchovesicular respiration; small mucous râles; vocal resonance increased	Lung tissue relaxed; loss of normal tension. Moderate atelectasis; œdema; deep congestion.
Diminished respiratory excursus on affected side or locally	Vocal fremitus increased	Dull.....	Bronchial respiration; increased vocal resonance; bronchophony	Lung tissue consolidated by compression, complete atelectasis or exudate; pneumonia; tuberculosis.
Diminished movement on affected side	Vocal fremitus diminished or absent	Flat.....	Absent respiration; sometimes distant bronchial breathing; absent voice; ægophony	Pleural effusion or tumor.
Respiratory movement restricted generally or locally	Vocal fremitus diminished	Vesiculotympanitic; tympanitic	Respiration feeble or cavernous; vocal resonance feeble or cavernous	Air increased within the vesicles; local or general emphysema or at particular points; cavities.
Diminished excursus over lesion	Vocal fremitus diminished	Amphoric; metallic; cracked-pot sound	Respiration amphoric or metallic; cavernous; amphoric or metallic voice; whispering pectoriloquy	Large cavity with elastic walls communicating with a bronchus.
Greatly diminished movement	Absent vocal fremitus....	Tympanitic; metallic; amphoric	Respiration metallic, amphoric; vocal resonance the same	Air in pleural sac; open pneumothorax.
		Coin test.....	Absent respiration.....	Closed pneumothorax.
		Dulness.....	Absent respiration; faint metallic voice	Air under great tension.
Greatly diminished movement	Absent fremitus; succussion	Flatness below. Tympany. Amphoric resonance above	Respiration and vocal resonance as above. Succussion sound; metallic tinkling	Air and fluid in pleural sac; hydro-pyo-hæmo-pneumothorax.

sounds of a feeble heart. The heart sounds should also be studied with full held inspiration and forced expiration.

Auscultation has for its object the determination of the character, intensity, and rhythm of the normal heart sounds, and their modifications within the range of health, the recognition of modifications which transcend those limits, and the detection of abnormal or adventitious sounds.

THE SIGNS IN HEALTH.

The Normal Heart Sounds.—When the stethoscope is placed over the heart two sounds are heard. Of these one is found to correspond in time with the cardiac impulse, and the other to follow it after a short interval of silence. After a longer, but still short interval, these sounds are repeated in the same order. For this reason they are spoken of respectively as the first and second sound of the heart.

The Characters of the Sounds.—The first sound is not only comparatively long, but it is also low in pitch and muffled. The second sound, on the contrary, is comparatively short and is high pitched and clear. The two sounds are therefore in sharp contrast in regard to their duration, pitch, and quality. The respective characters of the two sounds may be roughly imitated by the repetition of the syllables "*ubb dup.*"

Causes of the First Sound. — The first sound is due to vibrations caused by the simultaneous tension of the mitral and tricuspid valves in closure, the muscular contraction of the ventricles and the vibration of the blood contained within the ventricles at the moment of systole.

The Cause of the Second Sound.—The second sound is due to the vibrations caused by the simultaneous closure of the semilunar valves of the pulmonary artery and the aorta at the beginning of the ventricular diastole.

A Third Sound of the Heart.—Gibson has recently described a wave in the jugular pulse in healthy young adults occurring after the closure of the semilunar valves and before the auricular contraction, and accompanied by a low-pitched, clear sound at the apex, more distinctly audible in the cardiac revolutions which occur in the intervals between expiration and inspiration than at any other stages of respiration. This sound is not easily appreciated and is only audible in a certain proportion of the diastolic periods. It corresponds in time to the first element of the reduplicated second sound heard only at the apex, long familiar to clinicians. The explanations of this sound are at present purely hypothetical.

The Cardiac Cycle or Revolution.—Each revolution of the heart consists of an auricular systole, the instantly succeeding ventricular systole, and a period of repose of the whole heart. The relative time occupied with these events varies with the frequency of the action of the heart. With a pulse-rate of 74, that is, a cardiac revolution of about 0.8 second, the cardiac revolution comprises an auricular systole of 0.1 second, a ventricular systole of 0.3 second, and a period of repose of the whole heart of 0.4 second. With increased pulse-frequency the diastole of the ventricles is shortened much more than the systole; it is also, with slowing of the pulse-rate, lengthened to a greater extent. The statements which

assign regular proportions to the duration of the sounds and silences of the heart are misleading, since these vary in length, not relatively with varying heart frequency, but absolutely, the second silence being very fluctuating, since it corresponds to the fluctuating ventricular diastole, while the first sound and the short first silence together, which nearly correspond to the ventricular systole, are much more constant.

The Valve Areas or Puncta Maxima.—With the stethoscope applied over the apex of the heart the first sound is heard much more distinctly than the second sound and has a booming character which is in sharp contrast with the short and "valvular" quality of the latter. The temptation to rely upon the rhythm of the sounds for the recognition of the systolic sound or the first and second sounds is to be avoided. The auscultatory sign must be verified by inspection or palpation. This is especially important in the rapidly acting heart and in all morbid conditions. The systolic or first sound corresponds to the impulse as determined by sight or touch, or in default of these by the pulsation of the carotid. The radial pulse cannot be depended upon as a guide. The recognition of the first and second sounds is of especial importance in the diagnosis of valvular diseases.

When the stethoscope is carried to the base of the heart, either to the right or the left border of the sternum, the first sound becomes less distinct than at the apex while retaining its acoustic properties, and the second sound more distinct and prominent with an intensification of its snapping or valvular quality.

The sounds may be further analyzed by placing the stethoscope at the following principal points or areas:

1. **The Mitral Area.**—At or above the apex in the fifth intercostal space and upon the parasternal line. At this point that factor of the first sound made up by the closure of the mitral valve and the contraction of the left ventricle is best heard.

2. **The Tricuspid Area.**—At the juncture of the ensiform cartilage with the sternum and at the right border of the base of the sternum. In this region that factor of the first sound caused by the closure of the tricuspid valve and the contraction of the right ventricle is most distinctly heard.

3. **The Aortic Area.**—In the second right intercostal space near the sternum or directly over the second right costal cartilage at its sternal articulation—the aortic cartilage. At this point the aortic element of the second sound is best heard.

4. **The Pulmonary Area.**—In the second left intercostal space near the sternal border. At this point the pulmonary element of the second sound is best appreciated.

These areas do not correspond to the position of the respective valve systems, but they do correspond to the anatomical relationship to the wall of the chest of the structure in which the mechanism producing the sound exists, or in which the sound is conducted. That is to say, the anatomical apex of the heart formed by the left ventricle comes nearest to the chest at the apex; the tricuspid valve system at the right border and base of the sternum; the aorta just above its origin at the second right interspace, and the pulmonary artery above its valves at the second left interspace.

At the apex the first sound and its modifications in health and disease are best studied; at the base the second sound. In the former position this first sound is louder and more distinct; in the latter the rhythm is changed and the stress falls upon the second sound. The rhythm is the same in the mitral and the tricuspid areas and the quality of the first sound is similar, though in health the first sound is usually less intense in the tricuspid area. The rhythm is likewise the same in the aortic and the pulmonary areas, and the quality of the second sound is similar upon the right and left sides.

Modifications in the Normal Heart Sounds.—Variations in character, intensity, and rhythm are to be considered. There are marked differences in the sounds in different individuals and in the same individual at different periods of life and under varying conditions of activity and emotion.

Character.—The heart in children is less covered by the lungs than in later life and the chest wall is far thinner and more elastic. It follows that the sounds though feeble are more distinctly heard. As the muscle is smaller and thinner the valvular element of the first sound is more in evidence, and as the frequency is greater the long pause is shortened so that the rhythm, which at birth has the characteristic tic-tac of the fetal heart, like the ticking of a watch, only gradually changes to that above described as occurring in later life.

Embryocardia is a common condition in which the rhythm suggests that of the fetal heart, the long pause being shortened and the first and second sound presenting nearly the same acoustic properties. This modification of the cardiac rhythm occurs in tachycardia, the cardiac asthenia of the later periods of exhausting diseases and in extreme dilatation.

The first sound at the apex is not only somewhat louder in powerful persons with well-developed muscles but it is also more prolonged than in feeble persons who lead sedentary lives—a difference due to an increase of the muscular factor entering into the production of the sound.

A similar increase in the duration and intensity of the first sound occurs under conditions of bodily exercise and mental excitement. Under these circumstances the sound is occasionally attended by curious metallic reverberations, the *cliquetis metallique* of the French.

Intensity.—In young persons with thin, elastic chest walls the sounds of the heart are louder and more distinct than in older persons, in whom the walls are thicker and the costal cartilages more rigid. Thick layers of subcutaneous fat may render the sounds faint and distant. The interposition of the thick edge of a voluminous lung may have the same effect. There are marked differences in the intensity of the sounds in repose and activity.

The First Sound at the Apex.—The first sound is louder and more distinct in the mitral area than in the tricuspid, but in young persons under conditions of excitement or after great muscular effort it may be heard with equal clearness and intensity over the whole front of the chest.

The Second Sound at the Base.—The peculiarity of the second sound is its valvular quality. Its intensity varies in health with the energy of the heart's action. It has been assumed that the intensity of the aortic sound under normal conditions is greater than that of the pulmonary second sound. Vierordt, however, in 1885 first called attention to the fact

that the relative intensity of these two components of the second sound varies at different periods of life, an observation that has been confirmed by other recent clinicians and especially by the investigations of Creighton in 1899. This observer found that in 90 per cent. of healthy children under ten years of age the pulmonic second sound is more intense than the aortic; in 66 per cent. between the tenth and twentieth years the pulmonic sound is more distinct; in about half in the following decade, and after the thirtieth year the proportion gradually declines until after sixty, when the aortic second sound is more intense—accentuated—in almost every case. It thus appears that the relative intensity of the two elements of the second sound depends upon the age of the individual, the sound in the pulmonary area being more intense in early, and that in the aortic area more intense in later life, while in middle life their intensity is much the same. Cabot suggests that "it is therefore far from true to suppose that we can obtain evidence of a pathological increase in the intensity of either of the sounds at the base of the heart simply by comparing it with the other." The difficulty lies in the failure on the part of the auscultator to recognize the difference between mere *loudness* or *intensity* which may be normal, and *accentuation*, which is a morbid physical sign.

In elderly persons the second sounds are frequently heard more distinctly in the third or fourth interspace than in the second.

Rhythm.—The derangements of rhythm which may occur in health are:

1. *Gallop Rhythm in which the Diastolic Pause is Shortened with the Addition of an Extra Sound of the Heart.*—The rhythm suggests the cadence of the footfall of a cantering horse. It is expressed by the repetition of the syllables "rat-ta-ta." The mechanism of its production is not clear.

G. Canby Robinson has summarized the results of recent studies of gallop rhythm as follows: "Gallop rhythm of the heart is a fairly frequent clinical phenomenon, and consists in the presence of a group of three cardiac tones, none of which are murmurs. It occurs under variable clinical conditions. That form of gallop rhythm which is best heard at the apex or over the central part of the precordium may be divided into the presystolic, protodiastolic, and mesodiastolic types, depending on whether the extra tone falls at the end, at the beginning, or in the middle of diastole. Each form is associated with a characteristic cardiogram. There are a number of factors which probably combine in various ways to produce the various forms of gallop rhythm.

"Presystolic gallop rhythm is heard in two classes of cases. It is heard in strongly acting hearts in which a muscle sound produced by a strongly acting, hypertrophied auricle is probably the cause of the extra tone; and it is also heard in weak, rapidly acting hearts at the height of acute febrile diseases, at which time there is possibly a delay in the conduction of the heart-beat from the auricles to the ventricles. Under these circumstances the sound produced during the contraction of the auricles becomes distinguishable from that produced during the contraction of the ventricles. In both classes of cases, the extra tone seems to be produced in the auricle rather than in the ventricle. Protodiastolic and mesodiastolic gallop rhythm are caused by the production of an extra tone in the ven-

tricles. The factors that probably combine to produce this extra tone are an increase in the amount and velocity of the flow of blood from the auricles into the empty ventricles and a loss of tone of the heart muscle of the ventricles. The longer silent period in cases of gallop rhythm does not as a rule occur during diastole, but is usually a systolic silence."

This derangement of the cardiac rhythm may sometimes be observed in the normal heart when rapidly acting under conditions of great exertion or excitement.

2. *Reduplication of the Second Sound at the Base of the Heart.*—Splitting of the second sound may be heard at the base of the heart at the end of full inspiration, especially if the breath be held or after active muscular exertion. Its mechanism probably consists in the asynchronous closure of the aortic and pulmonary valve systems as the result of heightened pressure in the pulmonary circuit.

3. *Reduplication of the First Sound at the Apex.*—An impure first sound may occasionally be heard at the apex, especially at the end of expiration under normal circumstances. This modification varies from a mere blur or prolongation of the sound to a distinct repetition, constituting a form of the gallop rhythm. It may be represented by the syllables "trupp" or "turrupp." In health it is not constant in the same individual. It has been attributed to conditions temporarily giving rise to an increase in the *vis-a-fronte* of one or the other ventricle.

MODIFICATIONS OF THE HEART SOUNDS IN DISEASE.

Variations in the character, intensity, and rhythm which transcend the borders of health, together with wholly abnormal or adventitious sounds, are to be considered.

Character.—The acoustic properties of the heart sounds are modified not only by changes in the heart itself and in the arteries but also by pathological conditions in the adjacent parts and the state of the chest walls as regards elasticity and thickness. Finally the character of the heart sounds is modified by constitutional conditions. Changes in character are commonly associated with changes in intensity, but it is well for the student to train himself to appreciate modifications of character and of intensity as constituting distinct groups of physical signs.

1. **The Heart.**—The first sound is prolonged and dull in hypertrophy; when the associated dilatation is marked it is sometimes very clear and sharp. A metallic clinking—*tintement metallique*—is occasionally heard to the right of the apex-beat. The second sound is loud and distinct, often ringing in character and doubled. When valvular lesions are present the sounds are greatly modified and replaced or accompanied by murmurs.

In hypertrophy of the right ventricle the first sound at the lower part of the sternum is louder and fuller than normal; but with much associated dilatation it is clearer and sharper. Accentuation of the pulmonary second sound is frequently present.

In dilatation the first sound is shorter and sharper, in other words, more valvular in character than normal. The muscular element is diminished. With progressive thinning of the walls these changes become more

marked. The second sound when heard in aortic insufficiency may be distinct, or faint and obscure; when there is dilatation of the aortic arch it may be ringing and prolonged.

The second sound is rarely heard in the aortic area in aortic stenosis for the reason that the deformity of the cusps is such as to prevent their free play.

Accentuation of the pulmonary second sound is an important sign in mitral insufficiency.

The first sound is unusually sharp and clear in mitral stenosis, while the second sound in the second left interspace is strongly accentuated and sometimes reduplicated.

2. **The Arteries.** — Accentuation of the aortic second sound occurs in arteriosclerosis, — especially that form which accompanies chronic nephritis,—in atheroma and dilatation of the aortic arch, and in aortic aneurism. It is the sign of increased arterial tension and is associated with hypertrophy of the left ventricle.

3. **Diseases of Neighboring Organs.**—In pericardial effusion the heart sounds are not only indistinct but they also have a peculiar muffled and distant quality, due to diffusion. Accentuation of the pulmonary second sound is frequently an early and persistent sign.

In some cases of pneumothorax the heart sounds acquire a metallic quality; in pneumopericardium they are feeble, distant, and muffled.

They are distant and muffled in pulmonary emphysema, well transmitted in consolidation of the lung and in chronic interstitial pneumonia and pulmonary phthisis, and sharp and ringing during cardiac overaction, especially in young persons and in the periods of excitement and palpitation which occur in exophthalmic goitre, chlorosis, and anæmic states.

4. **Different Conditions of the Walls of the Chest.**—As in health so in disease, remarkable differences in the heart sounds occur as the result of differences in the chest wall. Through the thin and elastic tissues of the young the sounds are conducted with great distinctness; they are faint and diffuse when the chest walls are thick and fat, and when the cartilages are calcified, the sternum thickened, or when deformities of the chest derange the normal relation of the heart to the wall, or finally when a new growth is interposed.

5. **Constitutional Conditions.** — The first sound is shortened as well as faint in conditions of general asthenia such as result from actual starvation and wasting diseases. In enteric fever the first sound becomes progressively shorter, more indistinct and valvular in quality—a change due to the progressive wasting of the myocardium.

Intensity.—The significance of increase or decrease in the intensity of the heart sounds as morbid physical signs has already to some extent been indicated. It is important to note that as a rule increase in the intensity of the first sound is associated with its prolongation, while decrease in intensity is attended with decrease in duration. The loud first sound is in strong contrast with the short second sound; the faint first sound resembles it. As the feeble heart is commonly also a rapid heart, in which the long pause is shortened, it may become difficult to tell which is the first and which the second sound. The first sound corresponds to the impulse at the apex or to the carotid pulse.

Accentuation. — It is important at this point to emphasize the distinction between "loudness" and "accentuation"—a matter not always made clear in the books. Loudness or sound intensity has to do with the volume of a given sound; accentuation is that acoustic property which indicates suddenness in the application of the energy by which the sound is produced. The first sound of the heart is often loud, even booming, but never, according to my belief, accentuated. It may have a slapping quality as in mitral stenosis, but that is something altogether different from accentuation. The second sound of the heart at the base may be loud and distinct without being accentuated. It may become accentuated without becoming louder. Accentuation is then something quite different from loudness. The word conveys the idea of suddenness, sharpness, a certain vibrating quality due to quick and sharp tension. Loudness is a matter of degree; accentuation a matter of quality. From this point of view accentuation becomes a physical sign of great importance.

The first sound is increased in intensity in conditions which cause the heart to act with unusual energy. In intense emotional states the first sound is greatly increased and may sometimes be heard all over the chest. Such overaction may be pathological, as in mania and acute febrile states. The first sound is louder than normal in hypertrophy of the left ventricle, but less constantly so than has been assumed; even with a considerable degree of associated dilatation the sound may still be quite intense.

The first sound is enfeebled in conditions of general asthenia such as result from starvation, long-continued fevers, wasting diseases, hemorrhage, shock, and profound exhaustion from over-exertion; in dilatation of the ventricles, myocarditis, fatty heart, and rupture of the compensation in chronic valvular disease; in chlorosis and anæmia and in all conditions that interfere with its transmission to the ear of the auscultator, such as fat in the chest walls, emphysema, pleural and pericardial effusions, and certain mediastinal tumors. In conditions in which direct pressure is exerted upon the wall of the heart by effusion or tumor, its action is impeded and its sound enfeebled.

The second sound is increased in intensity in nervous overaction of the heart and in all conditions in which the lungs are retracted so as to bring the aortic arch and the conus arteriosus into more extended relation with the wall of the thorax. An apparent increase in the loudness of one or the other elements of the second sound is produced by the retraction of the anterior border of the lung upon the corresponding side. The second sound is diminished in intensity by those conditions, both general and cardiac, which weaken the action of the heart and diminish the intensity of the first sound.

The significance of changes in the intensity of the aortic and pulmonary elements in the second sound demands consideration.

It has already been pointed out that in normal individuals after middle life the aortic second sound is more intense than the pulmonary. A mere increase in the volume of the sound may be the result of increased cardiac action. An increase associated with that change of quality designated by the term accentuation constitutes a morbid physical sign and becomes more significant in proportion as the accentuation becomes more marked.

Accentuation of the aortic second sound occurs in all conditions in which the arterial blood-pressure — *vis-a-fronte* — is increased, namely, arteriosclerosis, chronic nephritis, and in aortic aneurism and dilatation of the aortic arch. In conditions characterized by habitual increase in arterial tension there is usually cardiac hypertrophy.

Diminution in the intensity of the aortic second sound occurs in conditions in which the blood thrown into the aorta by the ventricular systole is reduced in amount as in aortic and mitral stenosis and to some degree also in mitral insufficiency. Under these circumstances the aortic second sound may be so diminished as to be no longer heard at the apex. Weakening of the wall of the heart, as in fibrous and interstitial myocarditis, fatty degeneration, and extreme dilatation, likewise gives rise to enfeeblement of the aortic second sound. Relaxation of the peripheral arteries produces the same effect. The aortic second sound is extremely faint in collapse from any cause.

The pulmonic second sound is louder than the aortic in children and in young adults. A pathological increase in the loudness of this sound has the same significance in regard to the pulmonary circulation that an increase in the aortic second sound has in regard to the general circulation, namely, an augmentation in the resistance to the flow of the blood. This occurs in chronic valvular disease of the heart, especially in mitral stenosis and insufficiency, and in various pulmonary diseases, particularly emphysema, chronic bronchitis, phthisis, interstitial pneumonia, and compression atelectasis. These conditions are associated with hypertrophy of the right ventricle, compensatory in nature; when the compensation fails, the pulmonary second sound becomes faint and indistinct. Under all these conditions the more intense pulmonary second sound is also accentuated.

Weakening of the pulmonary second sound is the sign of a weakened right ventricle or tricuspid insufficiency. This sign is of great value in pneumonia as indicating failure of the right ventricle. The pulmonic second sound should therefore be systematically studied, since it affords at once indications for treatment and data for prognosis.

Rhythm.—Allorrhythmia is the general term used to designate deviations from the normal rhythm of the heart. See Arrhythmia, p. 468 *et seq.* under Radial Pulse.

Theories of the Mechanism of the Heart.—The neurogenic or older theory was based upon the assumption that the rhythmic contractions of the heart muscle are due to stimuli originating in the nervous system, either in the motor cells of the brain or, as modified by Volkman, in the cardiac ganglia. The myogenic theory, which we owe to the brilliant work of Gaskell and Englemann, is of recent origin and ascribes the contractions to automatic impulses originating in and conducted by the muscle. The subsequent studies of His, Keith, Tawara, Erlanger and others have shown that these properties are possessed not by the myocardium as a whole but by certain highly specialized muscle fibres embryonic in type, which differ in structure from the muscle fibres of the adult heart and are present in the remains of the primitive cardiac tube. These muscle fibres constitute a system beginning at the junction of the superior vena cava

and the auricle as a small net-like mass recently described by Keith and Flask and now regarded as the seat of the primary impulses, continued in the right auricle as the atrio-ventricular or Tawara's node and passing thence into the septum and branching into both ventricles as the bundle of His. The myogenic theory during the short period since it was first formulated has aroused widespread interest among anatomists, physiologists and clinicians and been singularly productive in its influence upon biological investigation and practical medicine. Among other matters it has brought about an orderly classification of the cardiac arrhythmias and shed much light upon their significance.

Properties of the Heart Muscle.—The studies of Gaskell and Englemann have shown the following properties to be inherent in the myocardium and to constitute the basis of the physiology and clinical pathology of the heart: 1. *Stimulus production*—the capability of the heart to generate automatic rhythmic impulses. 2. *Excitability*—the ability to respond to an adequate stimulus by contraction. 3. *Conductivity*—the power of transmitting the impulse from one place in the heart to another. 4. *Contractility*—the capacity to react to more favorable conditions, in respect of rest, nutrition, temperature and so on, with stronger contractions. 5. *Tonicity*—the degree of contraction which the heart maintains during diastole, by virtue of which the total volume of the organ and the size of the cavities are less than if full muscular relaxation were to occur in diastole.

Two other properties of the myocardium are essential to the myogenic theory. The first is known as the *All or None Law.* It is the formulation of the fact that while a skeletal muscle reacts in proportion to the vigor of the stimulus, the heart has only one kind of response to all degrees of stimulation. A stimulus barely strong enough to cause a contraction will cause the most powerful contractions of which the heart is capable under given conditions at the moment. No increase of stimulation will increase the force of the contraction.

The second is the *Absence of Tetanus.* A skeletal muscle reacts to continuous rapid stimulation by continuous contraction, whereas the vertebrate heart responds with a single contraction and to continuous stimulation by a series of distinctly separate contractions. This is attributed to the refractory period, the reaction to the stimulus failing to occur until the effect of the previous contraction has ceased.

The influence of the vagus and the accelerators upon the action of the heart is important in connection with the myogenic theory. The rate of beats, the conductivity, the excitability and contractility are decreased by the influence of the vagus fibres and increased by that of the accelerator fibres. Englemann holds that there are separate nerve fibres which influence each of those functions and designates those which regulate the rate chronotropic; those which influence the conductivity dromotropic; those which modify the irritability bathmotropic and those which act upon the contractility inotropic. Finally, the influence of the elasticity and contractility of the blood-vessels upon the heart through the action of the vasomotor system enters largely into pathological considerations and has an important bearing upon diagnosis.

Methods.—Formerly the arterial pulse and apex beat were the guides to the study of the cardiac rhythm. The sphymograph and cardiograph added something to our knowledge but less than was hoped. Auscultation revealed the sounds and murmurs which attended the beginning and the end of the ventricular systole and recognizes ventricular contractions which fail to reach the superficial arteries as pulse beats. The defect of all of these methods is that they give no positive sign of the condition of the auricles. Tracings of the jugular pulse reveal the condition of the right auricle. The polygraph tells the whole story of the contractions of the auricles and ventricles, while the sphygmogram and cardiogram supply definite data in regard to the opening of the aortic valve system. That this story has been fully and finally interpreted cannot be said.

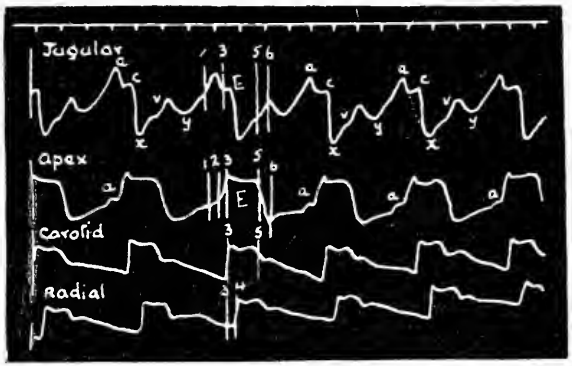


FIG. 81a.—Tracings of the jugular pulse beat, carotid and radial pulses. The perpendicular lines represent the time of the following events: 1, the beginning of the auricular systole; 2, the beginning of the ventricular systole; 3, the appearance of the pulse in the carotid; 4, the appearance of the pulse in the radial; 5, the closing of the semilunar valves; 6, the opening of the tricuspid valves. (MacKenzie.)

ABNORMAL OR ADVENTITIOUS SOUNDS.

Upon auscultation over the heart and great vessels sounds are heard in pathological conditions which differ from the normal heart sounds. These sounds bear a definite relation to the cardiac cycle and are dependent upon the action of the heart. Those which have their origin within the heart are spoken of as endocardial, those which arise outside of the heart as exocardial.

A. Endocardial adventitious sounds are called murmurs. They are:

Organic; Functional, Accidental or Hæmic.

B. Exocardial adventitious sounds, sometimes called pericardial murmurs, include:

Pericardial Friction; The Precordial Râles of Emphysema;
Pleuropericardial Friction; Pericardial Splashing;
Cardiopulmonary Murmurs; The Murmurs of Aneurism.

A. **Endocardial Murmurs.**—Much confusion has arisen from attempts to explain auscultatory phenomena in musical terms. Neither the sounds of the heart—sometimes erroneously called "tones"—nor cardiac murmurs, with exceptions presently to be mentioned, are musical phenomena. They both arise from irregular sound-producing vibrations which lack, as a rule, the rapidity necessary to the production of musical tones. A "sound" of the heart is produced by a single sudden derangement of the equipoise of sound-producing structures, which are thrown into vibration; a murmur by the continuous action of forces which maintain such vibrations. The sound presently

ceases; the murmur continues so long as the force which causes the vibrations continues to act. The sound corresponds in a way to a single blow upon a drum; the murmur to the continuous, rapidly repeated, but less intense sounds known as the roll of the drum; or the sound to the picking of the violin string, the murmur to the continuous note made by the drawing of the bow. But both these comparisons have the fault of likening musical phenomena to those which usually lack the musical quality. Furthermore the mechanism by which sounds and murmurs are produced is different.

The Mechanism of Endocardial Murmurs.—The heart sounds arise from the contraction of the heart muscle, the vibration of the blood mass, and the sudden tension of the auriculoventricular and semilunar valve systems. When murmurs arise a new set of physical conditions comes into play, namely, fluid veins (see p. 160). These swirls, or currents within currents of the blood, are attended with vibrations, which, first communicated to the wall of the heart or vessels and thence by way of the intervening tissues to the surface of the chest, are recognized by the auscultator as auditory phenomena—murmurs.

The Mechanism of Organic Murmurs—Lesions.—In by far the greater number of instances the fluid veins are due to actual lesions of the heart, and for this reason the murmurs are known as organic. The lesions mostly involve the valves, a fact which is indicated by the descriptive adjective valvular. They are on the one hand inflammatory and proliferative or adhesive, on the other sclerotic. Those that occur in early life are usually inflammatory; those which develop later are mostly sclerotic; but the inflammatory lesions of the valves undergo sclerotic changes, and old sclerotic valves are frequently the seat of recurrent inflammatory processes—recurrent endocarditis. As the result of each of these processes involving the valves, deformities arise. Inflammation causes vegetations, thickening, adhesions, and in extreme cases necrosis; sclerosis gives rise to thickening, retraction, crumpling; both result in loss of elasticity and freedom of movement. In cases of long standing lime salts are deposited and the rigidity and deformity are correspondingly increased.

Stenosis and Insufficiency.—The impairment of function is twofold. That function of the valves by which they yield before the blood stream and permit it to pass unhindered from auricle to ventricle or from ventricle to artery may be deranged. The condition is known as stenosis or narrowing, and the fluid veins are developed in the normal direction of the blood stream. Or that function by virtue of which the valves close their respective orifices is at fault, and there is valvular insufficiency or incompetency, the fluid veins developing in the reverse direction. Very often both these functions are impaired, and the condition is that of combined stenosis and insufficiency, with double murmurs.

Relative Insufficiency. — Again, the orifice guarded by a valve system may be enlarged in consequence of the dilatation of the heart, so that the edges of the valves may be unable to meet and close it. This condition is known as relative insufficiency or incompetence, and is dependent not upon lesions of the valves, but upon nutritive or degenerative lesions of the heart muscle. Acute relative insufficiency such as sometimes accompanies the heart failure of violent exertion is due to relaxation of the wall of the heart and papillary muscles.

Roughening of the surfaces of the valves or of the parts immediately adjacent to them and sudden dilatation of the artery just beyond the valve system may lead to the production of a murmur.

The deformity which gives rise to an endocardial murmur may be of all degrees, from such as only slightly impair the function of the valve system to a stenosis which leaves a tiny orifice or mere chink for the passage of the blood or an incompetence that is almost complete and transfers the pressure of the blood column to the wall of the chamber of the heart which is immediately behind the defective valve, namely, the left ventricle in aortic insufficiency and the left auricle in mitral insufficiency. A projecting firm vegetation or rigid spicule or the inelastic edge of a sclerotic valve may be the cause of a systolic murmur, where there is practically no actual narrowing of the orifice. One of the first lessons for the student of heart murmurs to learn is that by no means every systolic murmur having its point of maximum intensity in the aortic area is the sign of aortic stenosis.

Stenosis or Narrowing of an Orifice Guarded by a Valve System.—There is impairment of the function by which the valves open at the physiological moment. The flow of the blood is obstructed and under ordinary circumstances a murmur is produced, which is spoken of as an obstructive murmur. If the heart be very feeble, marked obstruction may exist without producing a murmur that can be recognized. If the left auriculoventricular orifice is involved, the condition is known as mitral stenosis or obstruction; if the aortic, as aortic stenosis or obstruction.

Incompetence or Insufficiency.—The function of the valves by which they close the orifice is impaired and a portion of the blood which has just passed through the orifice escapes from the main stream and flows back into the chamber of the heart whence it came. This pathological event is known as regurgitation, and the murmur which attends it is called a regurgitant murmur. We then have mitral and aortic incompetence, insufficiency or regurgitation as one or the other of these valve systems is affected.

Valvular lesions of the right side of the heart are of infrequent occurrence. They are sometimes the result of developmental defects or prenatal endocarditis. However produced they cause similar impairment of the valve functions, manifest by murmurs—tricuspid and pulmonary stenosis and incompetence. Stenosis is always due to deformity of the segments of a valve system. Incompetence is mostly due to the same cause, but not always. The deformity which prevents a valve from fully opening also generally prevents it from fully closing.

Combined stenosis and incompetence arises under the conditions just indicated. The lesion is a "double" one and manifests itself by a "double" or "to-and-fro" murmur.

Incompetence may, however, arise in the absence of stenosis as the result of (a) a lesion by which a valve segment has been destroyed by ulcerative endocarditis or has contracted adhesions to the wall of the heart, or (b) of relaxation of the cardiac muscle, as in relative insufficiency.

Stenosis without incompetence is comparatively infrequent; incompetence without stenosis is not very uncommon.

Valvular lesions exert their effect (a) upon the blood stream within the heart, (b) upon the walls of the heart, (c) upon the viscera, and finally (d) upon the peripheral circulation.

(a) The Effect of the Valvular Lesions which Produce Endocardial Murmurs upon the Blood Stream within the Heart.—The beginning of evil in stenosis and incompetence is the same. It consists in a reduction of the quantity of blood which eventually passes the diseased valve system with each revolution of the heart. In stenosis a portion of the stream corresponding to the extent of the pathological barrier is held back; in incompetence a portion corresponding to the degree of the pathological defect returns into the chamber whence it came—regurgitates. The result is a tendency to retardation of the flow, diminution in the volume of blood entering the arteries, and increase in the volume retained in the veins, with progressive transference of blood-pressure from the arterial to the venous side of the circulation. Were this tendency unchecked every case of valvular disease would in a short time terminate in death, the venous pressure rising and the arterial falling until the circulation becomes no longer possible. This result, which is the usual cause of death in valvular disease, is postponed for an indefinite period by compensatory changes in the muscle of the heart itself. It is true these changes are consecutive to the lesion, but as the latter is progressive, the former are correspondingly progressive. When the one advances at the same rate as the other a physiological balance is again established, the stability of which depends upon the tardiness of the valvular disease on the one hand, and the ability of the hypertrophied heart muscle to maintain its nutrition on the other. When extensive valvular defects develop suddenly or are rapidly progressive, compensation is not established and death occurs in a short time.

(b) The Effects upon the Walls of the Heart.—The immediate effects of the separation of the blood stream into a major part circulating under physiological conditions and a minor part held back under pathological influences are exerted upon the walls of the chamber behind the affected valve system. They are first dilatation, then hypertrophy. These changes may affect the whole organ, the heart acting as a single muscle and undergoing a general enlargement in response to the increased work required of it; more commonly they affect one or more of the chambers and in particular that chamber immediately subjected by the valvular lesion to increase in its blood contents in diastole and to a necessary increase in its energy in systole in order to overcome the obstacle in stenosis or propel an augmented volume of blood in incompetence. In mitral stenosis the left auricle cannot empty itself and receives blood from the pulmonary circuit; in mitral incompetence it receives blood at the same moment from the pulmonary circuit and the left ventricle; in combined mitral lesions some blood is retained and some regurgitates, while the physiological supply enters by the pulmonary veins. Consequently the left auricle is first dilated and then hypertrophied. In aortic stenosis the left ventricle cannot empty itself and in diastole the blood received from the left auricle is augmented by that retained at the time of the previous systole; in aortic incompetence blood enters the left ventricle in diastole

at the same moment from two sources, the left auricle and the aorta; in combined aortic disease the blood coming from the left auricle is augmented by that retained at the previous systole and that returning from the aorta. Under these circumstances the left ventricle is first dilated and then hypertrophied. It is important to bear in mind the cardinal fact that all these changes, like the lesion itself, are slight at first and gradually progress, and that in the early stages neither the dilatation nor the hypertrophy can be recognized by the ordinary methods of physical diagnosis. Even at this period the murmurs indicative of the respective lesions are commonly quite audible, often loud or harsh, and months may elapse before the signs of enlargement of the heart or venous stasis can be determined. The diagnosis rests upon the presence and characters of the murmur. Nevertheless it is an error to speak of this stage as preceding compensation. There are, however, cases of rapidly developing lesions in which complete compensation is only gradually attained; some in which it is never reached. If the quantity of blood held back in stenosis or regurgitated in incompetence be represented by x, it is evident that there must be an increased capacity of the affected chamber, represented by the same symbol, and that the chamber must be dilated to that extent. While if the resulting hypertrophy of the wall of the chamber be such as to enable it to propel the normal quantity of blood plus x, it is evident that a condition is established in respect to the volume of blood maintained in circulation, which is practically normal despite the valvular lesion, and this condition is known as compensation. This condition exists, however, by virtue of an abnormal increase in the nutrition and work of the heart muscle and at the expense of the normal reserve power of the heart, and is therefore unstable. It consists in a degree of dilatation and hypertrophy combined and in ratio to the valvular defect, but demands for its maintenance a hypertrophy slightly in excess of the dilatation. The nutrition of the overgrown and overworked muscle ultimately fails and dilatation develops in excess of hypertrophy. The compensation under these circumstances is said to be at first "deranged" or "failing," later "broken" or "ruptured." It is a question of degree. In a small proportion of the cases failure of compensation occurs in the absence of marked increase in the size of the affected chamber or chambers of the heart and has been ascribed to derangement of the innervation of the heart.

(c) Effects upon the Viscera.—Compensation, while adequate to the maintenance of a fair degree of health for an indefinite period, is never complete. There is always increased resistance to the onward flow of the arterial blood and a corresponding increase in the blood-pressure upon the venous side of the circulation. This results in increased fulness of the pulmonary circuit, manifest by accentuation of the pulmonary second sound, hypertrophy of the right ventricle and a *tendency* to passive hyperæmia of the viscera in general; hence, accentuation of the aortic second sound, dyspnœa upon exertion, a peculiar liability to bronchial catarrh and the occasional occurrence of blood-streaked sputum or slight hæmoptysis—phenomena which are common in mitral disease even while the compensation remains fairly good. Upon failure of compensation there is marked venous engorgement of the viscera, with grave derangement of

function, shown on the part of the lungs by marked dyspnœa or orthopnea, cyanosis, cough, and the occasional expectoration of frothy blood; of the liver and gastro-intestinal organs by loss of appetite, deficient digestion, nausea, slight jaundice and constipation; on the part of the kidneys by scanty urine and albuminuria. Dulness, stupor, somnolence with inability to sleep are symptoms of the derangement of the cerebral circulation.

(d) Effects upon the Peripheral Circulation.—While compensation is maintained, dropsy, as the result of valvular lesions, does not occur. There is sometimes to be found slight pretibial œdema, especially after fatiguing exertion or long standing. When compensation fails, however, the diminished arterial pressure and the increased venous pressure interfere with the circulation of the blood in the capillary zone and give rise to œdema. Under these circumstances, there is an accumulation of extravascular serum about the capillaries and a retardation of the lymph-flow. Hence the visceral derangements are not only hyperæmic, they are also œdematous. The action of gravity renders this accumulation of extravascular fluid early manifest in the dependent parts, namely, the legs and feet. As it increases, the thighs, genitalia, and loins become involved, and finally there is general œdema with effusion into the serous sacs. The dropsy of heart disease is often irregularly distributed, but its presence in particular localities may usually be explained by the relatively loose arrangement of the subcutaneous or other tissues involved, postural influences, and the action of gravity.

Compensation in mitral disease commonly fails by degrees, with periods of improvement following rest and treatment, and the ultimate catastrophe usually occurs after impaired health of prolonged duration.

Compensation in aortic disease is chiefly maintained by the left ventricle, which often becomes enormously hypertrophied, — *cor bovinum.* There is some increase in the venous pressure, since the ventricle receives its blood in diastole not only from the auricle but also from the aorta, but so long as the mitral valve remains competent, the visceral engorgement and general œdema which characterize the dyscrasia of the *stadium ultimum* of mitral disease do not occur. Precordial pain, angina pectoris, and momentary faintness upon rising or at stool are common, and in many cases the rupture of compensation is immediate and instantly fatal, death occurring with the heart in asystole.

The Mechanism of Functional, Accidental, or Hæmic Murmurs. — The murmurs designated by these terms are not signs of disease of the valves or orifices of the heart. The frequency of their occurrence enables us to determine with precision that they do not correspond to anatomical changes in the organ found upon examination after death. The mechanism by which they are produced has been the subject of much controversy, but none of the explanations advanced has been generally accepted. Functional murmurs are almost exclusively systolic and are heard over a limited space in the pulmonary area. They have been ascribed to dilatation of the conus arteriosus, to the fact that in anæmia and similar conditions there is lowered tonicity of the arterial walls which undergo an abnormal dilatation at the time of the systole, and to the lowered blood-pressure of anæmia in the aorta and pulmonary artery, which, in connec-

tion with the relatively high intraventricular pressure at the beginning of systole, prevents the closure of the semilunar valves and the systolic tension of the vessels, with the result that a systolic murmur arises upon the passage of the blood into the large vessels at the moment of physiological stenosis—contraction of the cardiac sphincters.

Diastolic functional murmurs, which are so rare that they scarcely demand consideration for diagnostic purposes, are probably venous murmurs transmitted to the innominata or vena cava and heard at the base of the heart.

The Significance of Endocardial Murmurs.—In order to determine the diagnostic meaning of a murmur heard upon auscultation of the precordial area, we must first answer the following questions: Is it endocardial or exocardial? (See page 185.) If endocardial is it organic or functional? (See page 196.) Having found it to be an endocardial murmur of organic origin we must then ascertain (1) its time in the cardiac cycle; (2) its point of maximum intensity; (3) the direction in which it is propagated and the area over which it can be heard; (4) its relation to the normal sounds of the heart; (5) its acoustic properties, and (6) the effects of exercise, respiration, and posture upon it.

(1) The Time of Murmurs in the Cardiac Cycle.—The determination of this point is of primary importance. For diagnostic purposes the systole may be regarded as lasting from the beginning of the first sound of the heart until the second sound; the diastole from the beginning of the second sound until the beginning of the first sound in the next revolution of the heart. A murmur heard at the time of the first sound or replacing the first sound or extending into or developing in the period between the first and the second sounds is *systolic*. Murmurs which develop in the latter period are designated *late systolic*.

A murmur which occurs at the time of the second sound or replaces it is *diastolic*. Murmurs which occur during the last portion of the diastole and run up to the first sound are known as *presystolic*.

When the heart is acting moderately there is no difficulty in recognizing the first and second sounds by their respective characters, and the long and short silences by their relative duration. But when the heart is rapid the different acoustic characters of the two sounds cannot always be made out and the rhythm is so deranged that the difference between the long and the short silence is less marked. Under these circumstances the systole may be determined by palpation with the finger over the apex or the carotid artery during auscultation, or by inspection if the double stethoscope is used. The interval between the time of the cardiac impulse and the radial pulse renders the latter a misleading guide for this purpose.

(2) The Point of Maximum Intensity of Murmurs.—The area in which a murmur is best heard is likewise of cardinal importance in diagnosis. The murmur is loudest at the point of its production and is best transmitted in the direction of the blood stream in which the fluid veins which produce it are developed. It is in accordance with these laws that a murmur having its point of greatest intensity in the mitral area has its origin at the mitral valve. It is necessary in this connection to bear in mind the fact that the mitral area is not constant in the normal position

but that it shifts with displacement of the heart. In a limited proportion of cases of mitral disease this murmur is best heard to the right of the normal position of the impulse, and in rare instances at the left border of the sternum higher up, even as high as the punctum maximum of the pulmonary second sound.

It is also in accordance with the above laws that murmurs heard in the pulmonary area, namely, the second left intercostal space, have their origin in the conus arteriosus or at the pulmonary orifice; that murmurs having their maximum intensity at the right lower border of the sternum, at the level of the fourth and fifth intercostal spaces, or at the base of the ensiform cartilage are produced at the tricuspid orifice, and that murmurs whose maximum intensity is in or near the aortic area have their origin at the aortic orifice. Murmurs arising at this point very often, however, are best heard over the upper part of the body of the sternum near its left border or, less frequently, at the apex or over the lower part of the sternum—aortic insufficiency.

(3) The Propagation of Murmurs and the Extent of the Area in which they can be Heard. — Murmurs are very often heard over limited areas and transmitted in definite directions. This is especially but not exclusively true of the murmurs produced by lesions of single valve systems. Thus the murmur of mitral stenosis—the presystolic murmur—is heard over a circumscribed area just above the apex and is not propagated, while the systolic murmur of mitral incompetence is heard over a considerable area to the right of the apex and upward and is transmitted distinctly in the direction of the left axilla and to the back. On the other hand the systolic murmur of aortic stenosis is usually prolonged and loud, heard over an extended area and transmitted into the carotid and subclavian artery; it is in some instances heard at a distance from the chest. The diastolic murmur of aortic incompetence may also be heard over an extensive area of the chest both in front and behind. A murmur distinctly heard over two or more valve areas may be due to one or to several valve lesions. If it is systolic in time, it may be the sign of mitral insufficiency or of aortic stenosis, or the murmur may be a compound of two murmurs, each representing one of these lesions. The difficulties are greatly increased when there are to-and-fro murmurs representing double lesions—stenosis and incompetence—of the respective valves. A correct diagnosis rests upon the application, in the study of individual cases, of the knowledge, obtained by clinical experience and post-mortem examination, that the murmur produced by each valvular lesion has its characteristic point of maximum intensity and definite line of propagation along which its intensity gradually diminishes as the stethoscope is moved away from that point. A murmur which fulfils these requirements in regard to a particular valve area and line of propagation and is unaccompanied by any other murmur may be regarded as the sign of a lesion of that valve. When, however, two or more murmurs are heard which differ in their acoustic characters, as pitch, quality, and duration, and present each its point of maximum intensity, and are propagated respectively in different directions, a correct diagnosis can only be reached by the separate study of each as though it alone were present, the others being for the time being

disregarded. In this analytical study too much importance cannot be placed upon the differences in pitch and quality and the evidences of the influence of the lesions which cause the murmurs, upon the size of the heart, the viscera and the pulmonary and peripheral circulation. In complicated cases of cardiac disease the diagnosis cannot be made by an analysis of the murmurs alone. A systematic study of the associated physical signs is essential to success.

When several murmurs are present, it is best not to attempt an over-refined diagnosis in regard to the lesions which underlie all of them. We may be content when we have determined with accuracy the two which are most important, and we may be reconciled to our inability to satisfactorily do more than this by the knowledge that greater nicety of diagnosis, though it might gratify our technical ambition, is not demanded by the requirements of therapeutics and prognosis, and has been discredited by the experience of the post-mortem room.

In this connection it is important to call the attention of the student to the fact that the intensity of a murmur does not necessarily gradually and progressively diminish in its line of propagation, but may be modified by the presence of a viscus or the interposition of a new growth. Thus an aortic murmur may be distinctly heard in the aortic area and near the apex and only faintly in the intervening space. This phenomenon has been attributed to the interposition of the right ventricle, while the transmission of a mitral regurgitant in the direction of the left axilla may be abruptly interrupted by a pleural effusion or neoplasm.

(4) The Relation of Murmurs to the Sounds of the Heart.—A murmur may accompany the sounds of the heart or may replace them. The systolic apex murmur of mitral incompetence wholly or in part replaces the first sound. The systolic basic murmur of aortic stenosis accompanies the first sound, but when compensation fails the first sound is greatly weakened, and with enfeeblement and dilatation of the ventricle or upon the supervention of relative mitral incompetence it may no longer be heard. In aortic stenosis the second sound is not often heard at the aortic cartilage, because the deformity of the valve usually prevents its closure. In aortic incompetence, the second sound may be well heard or it may be replaced by the murmur. In some cases it may be absent in the aortic area but heard over the carotid artery.

A murmur which accompanies a sound also follows it, since the time of the murmur is longer than that of the sound. A murmur may run up to a sound, as the presystolic murmur of mitral stenosis. In rare cases murmurs occur between the sounds. The persistence of the sound along with the murmur may be of favorable prognostic significance, as, for example, in aortic incompetence, where it indicates partial closure of the damaged valve cusps with corresponding preservation of function.

(5) The Acoustic Properties of Murmurs.—Under this caption the (a) intensity, (b) quality, (c) pitch, and (d) duration of murmurs are to be considered.

(a) *Intensity.*—The intensity of cardiac murmurs is extremely variable. A murmur may be so loud that it may be heard at a distance of some feet, or so low as to be scarcely audible when the patient holds his breath.

Not infrequently a loud murmur is heard by the patient himself. Such very loud murmurs are rare. The intensity of a murmur is by no means proportionate to the gravity of the lesion by which it is produced. On the contrary, since its intensity depends upon the energy with which the blood is propelled through the affected orifice, that is, upon the compensation, a loud murmur is, other things being equal, more favorable than a faint one. As compensation fails, the murmur becomes fainter and it not infrequently happens that in patients coming under observation with greatly impaired compensation no murmur can be recognized upon careful auscultation, but after rest and suitable treatment have brought about inprovement in the general condition and in compensation a murmur appears which becomes more intense as the patient grows better. This is especially the case in mitral disease. There are, however, cases of acute rheumatic endocarditis, especially in children, and of malignant endocarditis in which the changes in the valvular lesions develop rapidly while the power of the myocardium is still maintained, in which increasing loudness of the murmur constitutes a most unfavorable sign. The intensity of an organic, endocardial murmur is important less from its degree at any one time than from its decrease or increase during the progress of the case.

A murmur is not usually of the same intensity during its brief course. In general it is louder at the beginning than at the end. Presystolic murmurs are, however, usually louder at the close. The cause of the increase in intensity is here due to the fact that the blood flows gently through the auriculoventricular orifice at the beginning of the ventricular diastole, but with increased force under the stress of the auricular contraction later.

(b) *Quality.*—Endocardial murmurs vary in quality from a soft blowing sound—bellows murmur, *souffle*—of little intensity, to a coarse, harsh, rasping sound of considerable loudness. In rare instances they are musical. The musical quality is usually manifest during a part of the murmur only, the remainder having the ordinary blowing or rasping character. The musical quality indicates an organic lesion, but does not enable us to define its nature and is without significance in prognosis except that it indicates a certain degree of power in the heart muscle. The presystolic murmurs which are produced by mitral and tricuspid stenosis and the "Flint murmur" of aortic insufficiency have a peculiar "rumbling" or "blubbering" quality not heard under other conditions. These murmurs have been compared to a short roll of the drum, but they are much less regular.

(c) *Pitch.*—This attribute of murmurs is also variable. Blowing murmurs of soft quality are commonly low pitched, while the coarser murmurs are often high in pitch: to this general statement the exception that the very coarse, blubbering presystolic murmurs are usually of low pitch. It is the high-pitched murmur that tends to assume the musical quality.

(d) *Duration.*—A murmur may occupy the whole period of the systole or the diastole or any part of either of these periods. The systolic murmur of mitral incompetence is sometimes prolonged, the diastolic murmur of aortic incompetence almost always so. Presystolic murmurs are of shorter duration. The length of murmurs is not of itself of prognostic significance

(e) *Variation in Murmurs.*—Rapid changes in the acoustic properties of murmurs, even their disappearance and reappearance, may occur in acute endocarditis when vegetations are forming upon the valve segments, and especially in the malignant form, in which the vegetations grow with rapidity upon the valves and adjacent walls and ulcerative lesions occur. Similar changes may occur in the course of acute endocarditis as the result of rupture of chordæ tendineæ or the formation of adhesions between valve segments or between a segment and the wall of the heart.

(6) Effects of Exercise, Respiration, and Posture upon Endocardial Murmurs.—Faint murmurs usually become more distinct upon moderate exercise, as walking rapidly two or three times across a room, or stooping and rising several times in succession, and these movements are sometimes necessary in the examination of doubtful cases. When, however, a murmur has grown indistinct or disappeared as the result of rupture of compensation, movement simply increases the cardiac dyscrasia.

Organic murmurs are usually more distinctly audible upon quiet breathing, or while respiration is momentarily suspended, and at the close of expiration, when a larger area of the heart is uncovered. In this respect they differ from functional murmurs, which are frequently best heard upon inspiration.

Posture exerts an important influence upon the intensity of certain murmurs. Systolic murmurs not heard in the upright position may be distinctly audible in recumbency; on the other hand, murmurs not heard in the recumbent posture may be recognized when, by the patient leaning forward, the heart is brought into closer relation with the wall of the thorax. Presystolic murmurs are sometimes much better heard in the erect than in the recumbent posture.

The Significance of Functional, Accidental, or Hæmic Murmurs.—A large proportion of endocardial murmurs, much larger than was formerly supposed, are not associated with anatomical cardiac lesions. Certainly murmurs are not rarely heard *intra vitam* in cases in which no corresponding valvular lesions are found *post mortem*. Systolic murmurs arising in conditions of cardiac asthenia from relaxation of the cardiac sphincter—relative incompetence—and having all the characters of incompetence from actual lesions at the mitral orifice, though often transient are not usually described as "functional." Short, whiffing, systolic murmurs, sometimes heard in the mitral area directly after violent or prolonged physical effort, are probably due to relative insufficiency resulting from acute dilatation. They disappear in the course of a little time.

Functional murmurs are almost always systolic in time. By far the greater number of them have their point of maximum intensity in the pulmonic area; occasionally only are they most distinctly heard in the aortic or mitral areas. They are commonly well heard to a little distance from the point of maximum intensity in all directions rapidly diminishing in loudness, and are not distinctly transmitted in a definite line, as is usual with organic murmurs. They are as a rule soft and blowing in character. A loud coarse murmur, whatever its other points of resemblance to functional murmurs, is likely to prove to be organic, especially when persistent. Functional murmurs are usually most distinct at the close of inspiration.

They are commonly transient and disappear when the condition with which they are associated improves. They are not associated with the signs of enlargement of the heart or with accentuation of the pulmonary second sound.

Functional murmurs are significant of the various forms of anæmia. For this reason they are spoken of as "hæmic murmurs." They occur in secondary anæmias, chlorosis, pernicious anæmia, leukæmia and Hodgkin's disease. A distinct, prolonged systolic murmur in the pulmonary area is common in chlorosis, and, in consequence of the retraction of the borders of the lungs, is frequently associated with a loud pulmonary second sound. In the *stadium ultimum* of pernicious anæmia the hæmic murmurs often disappear.

The differential diagnosis between organic and functional endocardial murmurs rests upon the following facts:

Organic murmurs occur at any period in the revolution of the heart; functional murmurs are practically always systolic. It becomes necessary, therefore, to contrast the characters of organic systolic murmurs with those of functional murmurs.

Systolic organic murmurs are usually well propagated in the case of mitral insufficiency toward the left axilla and to the back; in aortic stenosis, to the carotids and the subclavians, especially upon the right side. They are often soft and blowing, not rarely coarse and loud, sometimes musical. The point of maximum intensity corresponds to the respective mitral and aortic areas as above described (see page 169), and only in exceptional cases is to be located in the neighborhood of the pulmonary area. Organic murmurs, except in the case of relative insufficiency, are persistent, diminishing in intensity and ultimately disappearing only when the compensation fails and is finally ruptured. They are sooner or later associated with the signs of enlargement of the heart and increase of the blood-pressure in the veins, as accentuation of the pulmonic second sound, visceral engorgement and anasarca. The anamnesis commonly points to an acute infection, rheumatic fever, hard work and worry as causal factors.

Functional murmurs, on the other hand, are not propagated in definite directions; practically always soft and blowing, very exceptionally loud or coarse; never musical. Their point of maximum intensity is almost invariably in the pulmonic area. They are transient and not associated with the signs produced by the effects of valvular lesions, as manifest in retardation of a part of the blood stream; accentuation of the pulmonic second sound, enlargement or distention of the walls of the heart; visceral derangements—venous engorgement; or disorders of the peripheral circulation —dropsy. Anæmia is almost always present.

The rare diastolic functional murmur, so rare as to be unimportant in diagnosis, has been observed only in anæmia of very high grade and in association with a venous hum.

B. **Exocardial Adventitious Sounds.**—Morbid physical signs not having their origin within the heart are frequently heard upon auscultation in the precordial region. Important in themselves, they acquire additional diagnostic importance by reason of their occasional close resemblance to endocardial murmurs. Of these the following are the more important.

(a) **Pericardial Friction.** — The friction rub of dry or fibrinous pericarditis is heard upon auscultation as a grazing, creaking, or rasping sound occupying some part of the time of the cardiac revolution. It is sometimes systolic, sometimes diastolic, often to-and-fro, but rarely corresponds to the systole or diastole as endocardial murmurs do. It occurs irregularly and with momentary interruptions, commonly changes in character and time from one examination to another, and may appear, disappear, and appear again in the course of a few hours. Pericardial friction is usually best heard in the area of superficial cardiac dulness, but may be heard at any part of the precordial region and sometimes over the greater part of it. In some instances it is confined to the base of the heart. It is always, however, distinctly circumscribed and never propagated in any direction beyond the borders of the heart. It conveys the impression of being produced close to the ear and is intensified by the pressure of the stethoscope, which also increases the pain which is present. It is apt also to be increased when the patient inclines his body forward. It is little influenced by the respiratory movements, except that in some instances its area is slightly extended in expiration. Pericardial friction is frequently associated with endocardial murmurs, the signs occurring as the manifestations of an endopericarditis or the pericarditis developing in an individual already the subject of chronic valvular disease. Under these circumstances the friction sound is usually more conspicuous than the endocardial murmur and at times may mask it altogether.

The differential diagnosis between an endocardial murmur and a pericardial friction rests upon a critical analysis of the signs in the light of the history of the case.

(b) **Pleuropericardial Friction.**—Cases occasionally occur in which fibrinoid exudate upon that part of the pleura which is in relation with the pericardium gives rise to a friction sound having the cardiac rhythm, the roughened pleural surfaces being moved in apposition to each other by the movement of the heart. The differential diagnosis between pericardial and pleuropericardial friction rests upon the following facts:

The pleuropericardial friction is commonly heard in connection with a friction sound having also the respiratory rhythm—pleural friction. It is apt to be increased by forced respiratory movements and to be more distinct upon inspiration, whereas pericardial friction is best heard at the close of expiration. A positive diagnosis cannot always be made.

(c) **Cardiopulmonary Murmurs.**—Murmurs, hitherto known as cardiorespiratory, having the cardiac rhythm, are occasionally produced in the borders of the lung in relation with the heart by the traction or pulsion of the heart upon the lung tissue in systole or diastole. The murmurs are pulmonary but not respiratory, and are due to the sudden displacement of a certain volume of air from a mass of lung tissue confined by adhesions. They are most commonly heard near the apex of the heart and over the projection of the left lung which overlaps it, known as the lingula; less often under the left clavicle or about the angle of the left scapula. These murmurs are mostly systolic—traction murmurs; very rarely diastolic—pulsion murmurs—and are heard over circumscribed areas. They are much influenced by active respiration and cough. They

occur during inspiration and are scarcely, if at all, audible during expiration, a fact which is of importance in distinguishing them from endocardial murmurs, which are usually better heard when the breath is held in expiration and a larger cardiac surface left uncovered by the retracted lung. Cardiopulmonary murmurs have the soft, breezy quality of the vesicular murmur and suggest an inspiratory act broken by successive movements of the heart, which in point of fact they are. In rare instances these murmurs are accompanied by crepitant or subcrepitant râles. Their importance from the standpoint of the diagnostician consists in their superficial resemblance to endocardial murmurs, from which they may be differentiated without difficulty.

(d) **The Precordial Râles of Emphysema.**—In rare cases of emphysema, in consequence of the rupture of the walls of vesicles, air finds its way along the interstitial tissue to the root of the lung and thence to the connective tissue of the anterior mediastinum. The superficial cardiac dulness, if not previously obliterated by the borders of the emphysematous lung, disappears with weakening of the heart sounds and the occurrence of high-pitched metallic or crepitant râles which have the rhythm of the heart. These signs are to be differentiated by their acoustic properties from the tricuspid regurgitant murmurs, due to the dilatation of the right ventricle, so frequently heard in emphysema. They are also to be differentiated from the râles having the cardiac rhythm, which are heard in rare cases of infiltration of the lungs or cavity formation in the neighborhood of the heart, by the persistence in the latter of superficial cardiac dulness and the heart sounds, the character of the associated respiratory sounds, and by the fact of their occurrence in pulmonary emphysema.

(e) **Pericardial Splashing.**—In pneumohydro- or pneumopyo-pericardium there may be heard peculiar splashing sounds of metallic character similar to the succussion sounds of pneumohydrothorax, but having the cardiac rhythm. The heart sounds under these circumstances are usually feeble and distant. The cardiac dulness is in the recumbent posture replaced by an area of tympany, the borders of which shift with changes in the posture of the patient. These splashing sounds can under no circumstances be mistaken for murmurs, but they may closely resemble the splashing of the gastric contents sometimes produced by the movements of the heart, or that of pneumothorax or of a large vomica, from which they may, however, be distinguished by the gravity of the symptoms, the concomitant signs of pericardial perforation or inflammation, the examination of the patient when the stomach is empty, or a systematic routine examination of the lungs.

(f) **The Murmurs of Aneurisms.**—Aneurism of the thoracic aorta more commonly involves the ascending portion of the arch. On auscultation at the base of the heart or sometimes in a wide area there may be heard a systolic murmur, transmitted like the murmur of aortic stenosis in the direction of the aorta itself and the vessels of the neck. A diastolic murmur is sometimes also present. The latter is due to the reflux of blood into the sac, and may easily be mistaken for the murmur of aortic insufficiency with which it is not infrequently associated, as the manifestation of relative incompetence in consequence of the dilatation of the aorta or of valvular

deformities resulting from sclerotic changes. The differential diagnosis rests upon the presence or absence of the signs of aneurism, as determined by systematic inspection, palpation, percussion, and the anamnesis.

SOUNDS HEARD OVER THE PERIPHERAL VESSELS.

Auscultation of the Arteries. Normal Conditions.—When the stethoscope is lightly placed over the larger arteries where they run superficially, sounds may be heard which correspond to the sounds of the heart. These sounds are produced (a) in the heart and (b) in the arteries themselves.

The carotid may be studied at the angle of the jaw or at the inner border of the sternocleidomastoid muscle; the subclavian directly above the clavicle and external to the sternocleido muscle or directly below it, in the arm between the pectoralis major and the deltoid; the brachial at the inner border of the biceps or at the bend of the elbow, the arm being slightly flexed; the radial just above the wrist, and the femoral immediately below Poupart's ligament.

(a) The normal heart sounds are transmitted to some distance along the course of the main arteries at the root of the neck and may be heard in adults over the carotids and subclavians, more distinctly upon the right than upon the left side. In infancy and childhood only the second sound is thus transmitted, the first being very indistinct or wholly inaudible.

(b) A systolic sound, due to the sudden tension of the arterial walls, may in some cases be heard over the abdominal aorta and the femorals. In the majority of cases no sound is heard over these vessels nor over the smaller superficial arteries so long as the stethoscope is applied without pressure. When, however, some degree of pressure is exerted upon the wall of the artery by the rim of the stethoscope, a systolic murmur is produced, often intense and high-pitched, the so-called *compression murmur*. This murmur is due to sudden narrowing of the lumen of the vessel at the point of pressure with the production of fluid veins. If the pressure is increased to such a degree as to obliterate the lumen of the artery a systolic sound is produced by the increased force of the tension of the arterial wall—*pressure sound*. These phenomena are physiological and without other clinical significance than that which attends the risk of attaching erroneous importance to them.

(c) *Intracranial Murmurs.*—In children up to the sixth year there is sometimes to be heard upon auscultation over the cranium, and especially over the anterior fontanelle and in the parietal regions, a distinct systolic murmur, which apparently originates in the internal carotids from some unknown cause and is without diagnostic significance.

(d) *The Uterine Souffle.*—A soft blowing systolic murmur is heard over the pregnant uterus. It is first heard about the end of the sixteenth week and increases in frequency until the eighth month, after which it remains stationary. This murmur is subject to great variation as regards quality, intensity, rhythm, and point of maximum intensity. It is usually most distinct low down and upon one or the other side of the uterus, sometimes at the fundus, but very rarely over the entire uterine body. It is attributed to the circulation of the blood in the arteries of the uterine

wall. The diagnostic importance of this sign is impaired by the fact that a similar murmur is occasionally heard in chronic metritis, uterine myomata, and ovarian cysts.

Single or double murmurs corresponding in time to the fetal heart-beats are sometimes recognized in auscultation in pregnancy. They have in some instances been found to be associated with defects of development or endocardial lesions of the fetal heart—*cardiac souffle*. In other cases murmurs have originated in the umbilical cord—*funic murmurs*.

Auscultation of the Arteries. Pathological Conditions.—(a) It is obvious that abnormal sounds—murmurs—heard in the aorta will be transmitted into the carotids and subclavians. The systolic and less intensely the diastolic murmurs of lesions of the aortic valve system are transmitted along the course of these vessels.

(b) In any condition in which the pulse is quick—*pulsus celer*—the arteries may yield upon auscultation a systolic sound. This sign is sometimes present in fever and is common in aortic insufficiency, and may be heard over the radials as well as over arteries of larger calibre. In aortic insufficiency of high grade a double sound is sometimes heard over the femorals, the systolic dilatation and the diastolic contraction of the artery being alike attended with an audible sound. Systolic and diastolic sounds in the femoral artery have also been observed in pregnancy and in chronic lead poisoning.

(c) *Double Murmurs in the Arteries. Duroziez's Murmurs.*—In well-marked cases of expansile pulse in which the blood wave rises rapidly and rapidly recedes there may frequently be detected over the femoral or brachial artery at a certain point in the gradually increased pressure of the stethoscope a double—namely, systolic and diastolic—murmur. Some care is necessary to exert the degree of pressure under which this sign is best heard. It may be observed in aortic insufficiency, chlorosis, and other conditions in which there is well-marked *pulsus celer*.

(d) *Subclavian Murmurs.*—Systolic murmurs occurring independently of pressure by the stethoscope are common. Heard upon one side only when the attitude of the patient is unconstrained and the arms hanging at the sides, such a murmur is very suggestive of apex disease of the lung with pleural adhesions implicating the artery in its course. They are commonly louder upon inspiration, exceptionally upon expiration. Such murmurs are occasionally to be heard upon one or both sides in normal individuals, and there are those who are able to produce them at will by assuming certain attitudes, with fixation of the arms and the muscles of the upper part of the chest.

(e) *Thyroid Murmurs.*—Systolic murmurs are very common over the enlarged and tortuous arteries in goitre and especially in Graves's disease.

(f) *Murmurs in Local Arteriosclerosis in Superficial Arteries.*—Systolic murmurs due to this cause are occasionally observed. They are audible in some cases without pressure by the stethoscope; in others upon a minimum pressure. They are most common in the carotids.

Auscultation of the Veins. Normal Conditions.—In healthy individuals the blood flows in the veins without sound or murmur. In rare instances the occurrence of a venous hum constitutes an exception to this rule.

Auscultation of the Veins. Pathological Conditions.—(a) The venous pulse in tricuspid insufficiency may by sudden tension of the valves and walls of the jugular, and in particular the valves of the bulb, give rise to a systolic sound which is scarcely to be distinguished from the almost synchronous systolic carotid sound, except by the fact that it slightly precedes it.

(b) *Venous Hum—Nun's Murmur.*—This is the single venous murmur of practical diagnostic importance. Since the return flow of the venous blood is to all intents uninterrupted, venous murmurs are continuous.

The patient should assume the upright posture with the head straight. The stethoscope should be placed over the space between the sternal and clavicular portions of the sternocleidomastoid muscle without pressure. The murmur in question when present is heard as a peculiar, sometimes blowing, sometimes coarse and humming or again musical continuous sound, with rhythmical systolic, diastolic, and inspiratory intensifications. It is best heard upon the right side and diminishes in loudness or entirely disappears when the patient assumes the recumbent posture. Rotation of the face toward the opposite side increases the intensity of the sound. Pressure with the stethoscope at first increases then enfeebles the murmur until it wholly ceases and the systolic sound of the carotid is heard. In some cases a feeble murmur loses its continuous character and is perceived only at the moment of systole, diastole, or during inspiration. Under these circumstances the murmur becomes continuous upon light pressure with the stethoscope or if the head is rotated toward the opposite side—manœuvres which enable the diagnostician to distinguish the murmur from arterial and inspiratory murmurs. In other cases the diastolic portion of the murmur may be transmitted to the base of the heart and heard there as a diastolic murmur apparently of endocardial origin. The differential diagnosis may be attended with difficulty unless auscultation is practised from point to point from the heart along the course of the jugular, when it will become clearly apparent that the diastolic murmur heard over the base of the heart is in point of fact the transmitted venous hum. The venous hum occurs in anæmic and chlorotic persons and occasionally in healthy individuals with normal blood. In the last it has been assumed that the phenomenon is due to some anatomical peculiarity, as, for instance, sudden and unusual widening of the jugular at the bulb.

In general terms the causes of the venous hum are the more rapid flow of the blood current by reason of its lowered specific gravity and deficient hæmoglobin, and the sudden widening of the jugular at the bulb. The fact that this murmur is louder in the erect posture is rightly ascribed to the influence of gravity in hastening the flow; the increased intensity upon inspiration to the aspiration exerted by the chest movement at that time, and the greater loudness upon the right side to the more direct and unrestrained flow of the blood arising from differences in the anatomical arrangement of the veins of the two sides.

III.

THE EXAMINATION OF THE STOMACH AND INTESTINES.[1]

General Considerations.—Diseases of these organs are (a) primary or organic and (b) secondary or symptomatic, and in each of these groups there are cases in which recognizable anatomical lesions are present and cases in which there is merely derangement of function. Thus, carcinoma and ulcers are examples of organic disease with characteristic lesions, and hyperchlorhydria and pyloric spasm are functional affections; while loss of appetite, eructations, and vomiting occur as symptoms of phthisis, often without actual lesions of the stomach, and erosion and ulcer are not infrequent in chlorosis. It is of cardinal importance that the differential diagnosis between organic and functional disease of the stomach and intestines should in all cases be made, particularly as the former only require direct local treatment and the latter are frequently made worse by such treatment.

The Anamnesis.—It is very common for patients to attribute to "stomach trouble" or "bowel trouble" symptoms due to diseases of other organs or to constitutional disease. A careful and systematic anamnesis is therefore necessary in all cases.

Status Præsens.—The actual condition is ascertained by, (a) physical examination, with special modifications, as inflation, transillumination, the Röntgen rays; (b) chemical, and (c) microscopic examination of the gastric contents and alvine discharges.

EXAMINATION OF THE STOMACH.

(a) **Physical Examination.**—Inspection.—The neck, thorax and whole abdomen should be exposed, and the patient studied in the erect as well as in the recumbent posture. In the neck may sometimes be seen the enlarged left supra-clavicular lymph-node in cases of carcinoma of the subdiaphragmatic viscera. The general conformation of the thorax and abdomen, the type of epigastric angle and the number of floating ribs are studied, and in some cases the flaring of the left costal arch from the pressure of a distended high-placed stomach. When the abdominal wall is thin and relaxed the outline of the distended stomach, the slow, large movements of gastric peristalsis from left to right, far more rarely reverse peristalsis, the presence of tumor masses in the gastric wall or at the pylorus, or of metastatic growths about the umbilicus, in the liver, or elsewhere, may be observed. The normal pylorus is never visible.

Palpation.—This method is useful in determining localized or general tenderness, rigidity, the presence of a tumor, enlargements and displacements of the stomach, and "splashing." The stomach should be palpated systematically with both hands and for several minutes. By this means peristalsis may be aroused and a tumor which is not otherwise recognizable brought within reach. Under normal conditions the pylorus is not palpable, being

[1] Originally contributed by Dr. Gwyn.

separated from the anterior abdominal wall by the overlapping liver, when the stomach is empty. When it is prolapsed or the liver is small or in emaciated persons, the pylorus may be felt as an elongated, thickened mass the size of the thumb, extending obliquely to the right and upward somewhat above the level of the umbilicus beneath the right rectus muscle. It may be recognized by frequent rapid changes in its consistence and the palpable and sometimes audible forcible passage of the stomach contents through it from left to right. Succussion or "splashing" may be recognized both by the palpating hand and by the ear. It occurs in motor insufficiency and is of diagnostic value when elicited after a fasting period of several hours. The patient should be in the dorsal posture with the head and shoulders slightly elevated, and at the moment the examination is made the stomach should be depressed by deep-held inspiration. The examiner exerts pressure upon the epigastrium at the level of the xyphoid appendix

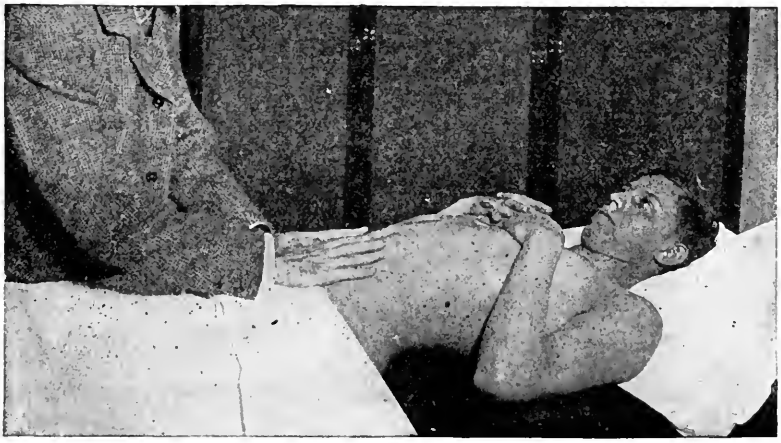


FIG. 82.—Palpating the abdomen.—Cohnheim.

and with the finger-tips of the other hand makes quick pressing movements immediately followed by relaxation.

PERCUSSION.—This method is of little value by itself. The stomach and adjacent coils of intestines yield tympanitic resonance or, when they contain fluid or solid matter, dulness, and their boundary lines cannot be defined by ordinary percussion. Auscultatory percussion, especially when the stomach is inflated, is of use. The percussion should be performed in lines radiating from a centre over the stomach and the points of change in the auscultatory phenomena marked upon each line in turn. These points are then connected. Control observations may be made by changing the centre and repeating the observation.

AUSCULTATION has only a limited application in the examination of the stomach. It is employed in the study of deglutition phenomena, "splashing," the *Spritzgeräusch* of pyloric stenosis and hour-glass contraction of the stomach. In the last, if the narrowing be of high degree, there may be an audible sound produced by the passage of water from the cardiac to the pyloric loculus, especially if pressure be used.

The Swallowing Test.—This test is used to determine the patency of the cardiac orifice and is performed in the following manner: The ear or the bell of a stethoscope is placed at the left of the tip of the ensiform cartilage or, better, at the left of the seventh thoracic vertebra of the patient, standing, who is directed to take a mouthful of water and swallow. There is at once heard the rumble of the contracting œsophageal and cervical muscles brought into play and in about seven seconds the trickling of the fluid entering the stomach through the cardiac orifice. Delay or absence of the latter sound suggests more or less complete stenosis in the course of the œsophagus or at the cardia.

Inflation.—The stomach may be distended by means of a stomach-tube and an ordinary rubber bulb syringe or by carbon dioxide evolved within the organ itself. To this end 3 to 5 grammes of tartaric acid dissolved in half a glass of water are swallowed and this is followed by an equal amount of sodium bicarbonate dissolved in the same quantity of water. The stomach first distends at its least resisting part and in the case of moderate enlargement and thin abdominal walls its greater curvature and inferior border may be made out by inspection or auscultatory percussion. The gas may be expelled as it is formed either at the cardia or at the pylorus, and in large stomachs the introduction of a sufficient quantity of air may cause much discomfort or even pain, or the gas evolved may be insufficient. The chief value of this method lies in the determination of the size, outline, and position of the stomach. It is of importance in the differentiation between tumors of the anterior wall and those situated in the posterior wall or behind the stomach. It is evident that upon inflation the former will become more prominent, the latter less obvious. Inflation is of some value also in bringing into prominence obscure tumors of the pylorus.

Position of Fluids Ingested under Observation.—The position of the lower border of the stomach is ascertained as closely as possible, the patient standing. He is then directed to swallow a glass of water and the resulting dulness is determined by percussion. This manœuvre being repeated two or three times at short intervals, changes in the level of the dulness may occur, which are indicative of the position of the lower border of the stomach. In normal stomachs of good musculature the increased amount of fluid enlarges the area of dulness in an upward direction; in relaxed and dilated stomachs the dulness sinks with the increasing weight. The method is of no great value except in dilated stomachs and gastroptosis in individuals with thinned abdominal walls.

Transillumination.—The gastrodiaphane of Einhorn consists of a flexible tube carrying at its top a small electric light. The Kemp instrument has the advantage in that its position can be controlled from the mouth. The patient drinks two or more glasses of water; the tube is introduced and the circuit completed. The light shows through the abdominal wall in the normal stomach as a triangular area having its apex and focus of intensity somewhat to the left of the median line and above the umbilicus; in gastroptosis or dilatation the point of illumination is lower and the light more diffused. Changes follow movements of the bulb. This procedure shows the lowest limit of the stomach at one point. There is no certainty

that the lamp does not push the greater curvature into positions it would not otherwise occupy, or that the position of the illuminated area affords positive data in regard to the size and shape of the organ.

Direct Gastroscopy.—Direct inspection of the mucous membrane of the stomach through a rigid metal tube requires the patient recumbent, with his head extended in such a manner that the trachea and œsophagus approach nearly a direct course. General anæsthesia is recommended for the best results. The tube used is fitted with a small electric light, mirror, and obturators, and in general resembles those employed in the diagnosis of rectal and sigmoid disorders, being, however, longer and slightly thinner. Many conditions of the stomach are readily recognized by direct inspection. Great care is necessary to ensure inspection of the whole interior, particularly if the stomach is enlarged. The cardia is first inspected. Inflation aids in bringing other parts into view. By some manipulation of the upper end of the gastroscope and simultaneous palpation and manipulation on the part of an assistant the whole stomach can be gone over and outlined. The readiness with which the end of the tube can be felt through the abdominal wall is of some service. The use of the œsophagoscope and gastroscope demands great technical skill and is not without danger.

The Röntgen Rays.—This method of examination has practically taken the lead among other devices for determining the form, size and position of the stomach. Röntgenoscopy and röntgenography are employed. Serial röntgenography yields the most satisfactory results in the examination of the gastro-intestinal tract. Plates sufficiently large to include the dome of the diaphragm above and the rectum below should be used. The efficient employment of this mode of examination requires not only elaborate and expensive apparatus but also great technical skill and should only be entrusted to those who devote their whole time to it as a specialty. The practitioner who attempts to do general work of this kind invites failure both in making the röntgenograms and in appreciating their significance. The patient should take castor oil the night preceding the exposures and no food having been eaten, he should swallow 350 to 400 grams of buttermilk with which a suspension of 100 grams of chemically pure barium sulphate has been stirred. The exposures follow immediately and after 1, 2, 5, 12, 18, 24, and 48 hours. The plates show the form, size and position of the stomach, the time required by the organ to empty itself and for the passage through the intestines, also the exact form-relations in various lesions, as ulcer, carcinoma, cicatrices, and so forth.

The fluoroscopic screen is very useful, in that the movements of the stomach can be watched and recorded.

The Stomach-Tube.—Various styles are in use. They are made of soft red rubber with a lumen of about .50 to .75 cm., walls not too thick, and about 70 to 90 cm. in length; near the gastric end one or two large lateral openings. Whether there should be an opening at the end is a matter of opinion. The upper end is fitted to a glass funnel of a capacity of 500 c.c. At the middle there may be a bulb which serves for inflation or suction and permits free siphonage. As the distance from the incisor teeth to the cardia is on the average 40 cm., or slightly less than 16 inches, there should be an encircling mark at this point. It is important to note that many of the tubes

supplied at the shops are marked at a point 51–60 cm. from the tip, about the distance to the lowest point of the greater curvature. For infants a soft catheter may be used.

Introducing the Tube.—The sitting position is easiest. It is best not to elevate the chin, since stretching the neck seems to occlude the upper œsophagus. Plates and false teeth should be removed. Soaking the last several inches of the tube in hot water makes the first contact of the tube with the pharynx less irritating. Holding the tube in the hand, as one would a pen, with five or six inches projecting, the examiner instructs the patient to open his mouth moderately wide, with the tongue touching and against the teeth. The tube is then passed straight back to the middle of the posterior pharyngeal wall and directed downward. If the patient can swallow at this moment the tube is usually engaged at once in the upper œsophagus and can be rapidly pushed in, reaching the stomach in several seconds, and is securely in place before the first expulsive coughing efforts begin. By ordering the

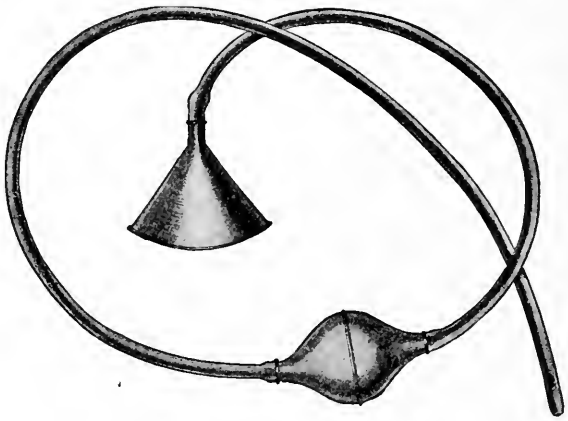


FIG. 83*a*.—Stomach-tube.—Cohnheim.

patient to breathe deeply several times one usually succeeds in quieting most of the discomfort and can proceed with the examination. To avoid the doubling up of the tube or the unpleasantness of the coughing or vomiting which often ensues, many prefer to introduce the forefinger of one hand along the side of the mouth as a guide and to pass the tube along this guiding finger. Many attempts are often necessary to overcome the spasmodic expulsive efforts of the pharynx. At times the distress is such that it is necessary to forego the attempt. Cocainization of the pharynx has been recommended; it does not, however, seem to have been very successful in difficult cases.

The contents of the stomach may be obtained by voluntary contractions of the abdominal wall, by the vomiting produced by slight movements of the tube or by aspiration. If aspiration is required, compression is made upon the bulb and the distal tube closed by grasping it between thumb and index finger of the other hand. The pressure on the bulb is removed and the contents are aspirated into the bulb, release the distal compression and allow the material to flow into a basin. Repeat the procedure as often as required. The stomach contents are usually removed one hour after the test meal when using this style of tube.

The great value of the stomach-tube in gastric diagnosis lies in the ease with which the stomach may be inflated and its contents removed. Much time will be saved by having the patient take one or other of the various test-meals whenever the tube is to be passed. After removal of the meal the patient's clothes are loosened and he is directed to lie down with the tube still in position. Inflation can now be performed and in conjunction with some of the various methods already mentioned will be found to be a very satisfactory way of estimating the size and position of the stomach.

The stomach is inflated until the patient indicates the beginning of discomfort. The examiner then clamps the tube or can ask the patient to hold it firmly in his teeth, thus giving the examiner the free use of both hands. During the inflation the examiner should carefully watch for the area where the stomach first manifests its presence. In a normal stomach this will be just below the left costal margin and in the epigastrium between the ensiform and navel. The stomach will stretch easily and its greater curvature can be followed to the umbilicus before overdistention is complained of. The lesser curvature must be outlined as well, either by inspection, percussion, or auscultatory percussion, and marked in pencil. Its position should be under the costal arch as high as the sixth and seventh ribs, and just below the ensiform cartilage in the midline. The fundus may distend high up toward the axilla. A distinct stomach-shaped outline can usually be obtained. It is only by outlining both lesser and greater curvatures that the difference between displaced and dilated stomachs is determined.

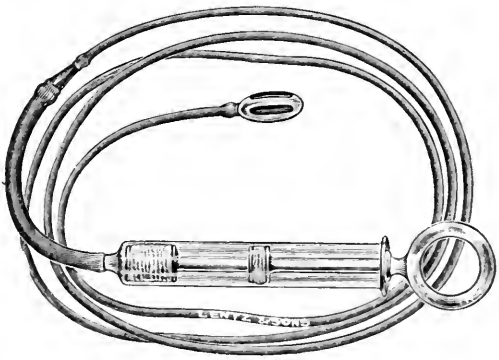


Fig. 83*b*.—Rehfuss stomach-tube.

The great advantage of the use of the stomach-tube in inflating is that the process can be repeated as often as may be desired without undue discomfort or delay, since after the first few minutes the patient experiences little or no uneasiness.

In addition to the estimation of the size, shape, and position of the stomach, the stomach-tube is used to determine conditions of hypersecretion and retention. To test for hypersecretion, the stomach-tube is passed in the morning before any fluid or food has been ingested, or the stomach may be washed out and the tube again passed after several hours' fasting. The recovery of more than 10 to 20 c.c. suggests disturbance of the gastric functions. In testing for retention the examiner washes the stomach clean, administers certain solid articles of diet, and passes the tube to obtain samples of the stomach contents, seven or eight hours later, or, according as marked conditions are suspected, twelve, twenty-four, or even forty-eight hours after the taking of the meal.

Contraindications for the Use of the Stomach-tube.—Those who for repeated diagnostic or therapeutic purposes have become accustomed to the

tube take it without difficulty and many learn to introduce it themselves. Its first introduction is often attended with great gagging, straining, and congestion, and is not wholly without danger in elderly persons with arteriosclerosis, myocarditis, and emphysema of high degree. It is also hazardous and unjustifiable in hemorrhagic cases, especially hæmoptysis, hæmatemesis, or cases of marked anæmia with tarry stools, and in aneurism of the aorta, in great debility from acute or chronic illness, and in pregnancy. Even in the absence of any of the foregoing conditions the retching, gagging, and distress of the patient may be so great that the attempt to pass the tube must be temporarily abandoned.

The fractional tube can be used in practically any condition. Rehfuss claims he has used it in all forms of gastric disease with no ill effects.

(b) **The Chemical Examination.**—The further examination of the stomach consists in the administration of certain test-meals or substances, their removal by means of the stomach-tube after a given period of time, and the application of various chemical tests for the digestive agents of the gastric juice.

Test-Meals.—Several standard test-meals are in use.

Plain water is a slight gastric stimulant and brings out a clear exudate from the gastric walls.

The Ewald Test-breakfast.—This consists of 35 grammes of stale bread or toast without butter and 200–400 c.c. of weak tea or water. Two small slices of toast without butter and one cup of weak tea without cream or sugar represent these amounts fairly well.

Riegel's Test-meal.—Two hundred c.c. mutton broth, 150–200 grammes beefsteak, potato purée 50 grammes, one roll 35–50 grammes is given to show the reaction of the stomach to a mixed meal.

Oatmeal meals are supposed to be free from lactic acid. Other meals have in addition urea or other chemicals in order to determine the amount of material which has been evacuated from the stomach.

Retention meals containing substances easily recognized—such as prunes, raspberries or lycopodium powder—are often given but are not necessary since the introduction of the fractional method of gastric analysis.

Starch.—The effect of the salivary enzymes can be first and most simply ascertained. The conversion of starch to achroödextrin and maltose goes on in the stomach until the free acids of gastric secretion reach a certain point. The well-known iodine reaction, coloring starch violet, coloring erythrodextrin, the first product, mahogany brown, and having no characteristic color effect in the final stages, achroödextrin and maltose, allows us to estimate quickly and qualitatively the extent of salivary digestion. Both the filtrate and residue contain reacting substances, soluble and insoluble starch. Excess of unaltered starch gives at once with iodine solutions (Lugol's solution .1 Gm. iodine, .2 Gm. potass. iodide, 200 c.c. water) a deep violet color; achroödextrin and maltose show no color reaction, though the latter is readily detected by Fehling's solution. Iodine, however, must be added in excess, since achroodextrin has a greater affinity for it than has starch, and the violet starch reaction may only appear after all the achroödextrin has been satisfied. The same color effects can be readily seen under the low power of the microscope.

The main relationship which starch tests, *i.e.*, salivary digestion, bear to stomach digestion is that hyperacid conditions of the stomach interfere with its progress, and that hypo-acid conditions may favor it.

The more important tests in ordinary clinical work are those for (1) acidity; (2) presence of free acids; (3) presence of free HCl, lactic and butyric acids; (4) presence of HCl in combination (combined HCl). Tests for proteid digestion, pepsin and peptone reaction, are usually considered to be unnecessary when normal or increased free HCl is found. Milk-curdling ferment is rarely tested for. A fat-splitting ferment in small amounts has been occasionally demonstrated, but is not regarded as of great practical importance. The qualitative tests in common use can be first considered.

Qualitative Tests.—1. Test for Acidity.—The products and agents of gastric digestion are normally acid, this reaction being due to free acids —HCl, lactic acid, butyric acid, and their combinations. Blue litmus paper is reddened by their presence.

2. Tests for the Presence of Free Acids.—Congo red paper or solution is turned deep blue by free HCl. A less intense reaction is given by the organic acids.

3. Tests for the Presence of Free HCl, Lactic Acid, etc.—*HCl.* (a)— *Methyl Violet Reaction.*—To a pale violet solution of methyl violet (one drop concentrated aqueous or alcoholic solution in a test-tube of water) add a few drops of the filtrate. A distinct blue change takes place if free HCl is present. A control tube should be on hand for comparison.

(b) *Tropæolin 00 Reaction.*—Two or more drops of fresh concentrated alcoholic solution of tropæolin (a deep orange-colored solution) are spread on a porcelain plate or dish. The same amount of filtrate is added to this surface and the porcelain gently heated. A distinct violet reaction turning to blue takes place.

(c) *Phloroglucin-vanillin (Günsburg's) Reaction.*—Two or three drops of the solution (phloroglucin 2 Gm., vanillin 1 Gm., alcohol 30 c.c.) are used with the same amount or more of the filtrate as in the tropæolin test and dried by gentle heat. The brown color of the phloroglucin-vanillin is changed to a distinct carmine red if free HCl is present.

(d) *Dimethylamidoazobenzol Reaction.*—A drop of a .5 per cent. alcoholic solution of this substance (a light red-brown solution) added to the filtrate or to the residue, quickly gives a bright red color if free HCl is present. In cases where but a few drops of gastric contents have been secured this test can be readily applied without waiting to filter.

The last two tests are by far the most reliable and are generally considered as absolute tests for the presence of free HCl. Lactic acid in excess may give suggestive results in the methyl violet and tropæolin reactions.

Lactic Acid.—Uffelman's Test.—One drop of a 10 per cent. solution of ferric chloride is added to 20 c.c. of a 1 per cent. solution of carbolic acid. The resulting deep blue mixture is diluted until it appears as a light amethyst. On the addition of a gastric filtrate containing lactic acid the amethyst changes to distinct yellow. Excess of free HCl, sugars, or peptones may decolorize the amethyst, and the yellow tint of many filtrates if added in excess gives suggestive but uncertain results. A comparison with a

test solution of lactic acid is always useful for a beginner. Far better results are obtained by shaking a portion of the filtrate with ether, which extracts the lactic acid, and applying the test to the evaporated residue, which may be preferably diluted with 2 or 3 c.c. of water. Strauss' modification of this test is also serviceable. One may dispense with the separator if it is not obtainable. Five c.c. of gastric juice are placed in a test-tube; 20 c.c. of ether are then added, the tube corked, thoroughly shaken for a few minutes, and allowed to settle. The overlying ether and extract can now be carefully removed with a pipette and mixed with 5 c.c. of distilled water. To this mixture two drops of a 1 in 9 watery solution of ferric chloride are added and the mixture is again shaken. The watery layer, as it settles below, is of an intense greenish-yellow color if lactic acid be present.

Butyric Acid.—This acid is usually only tested for by its odor. This and others of the volatile fatty acids, acetic and valerianic, are only looked for in marked conditions of stagnation of the gastric contents. Very minute quantities of them all, however, occur in various foodstuffs.

Combined HCl.—A qualitative test for the proteid combinations of HCl is not in general use. The quantitative tests will be considered below.

Proteid Digestive Power.—For the qualitative tests of the power of the gastric juice to digest proteid, one or two simple devices have been used. Fibrin and coagulated albumin (egg albumin) are the common proteids used: the fibrin well washed, hardened in alcohol and stained by neutral carmine, will digest in gastric juice containing free HCl and pepsin, imparting a red color to the liquid mixture as the carmine is set free by the digestion.

Small pieces, or disks, 2 mm. in diameter, 1 mm. in thickness, of not too firmly coagulated egg albumin are placed in a few c.c. of gastric contents. According to the amount of pepsin and free HCl present digestion begins more or less rapidly, and softening of the edges of the disks can be seen in one to two hours. Many hours are required for the complete granulation of either fibrin or albumin. Gastric juices deficient in free HCl have less and less effect upon the proteids employed. Some slight digestion goes on even with complete absence of free HCl.

Neither of the proteid digestive tests is very instructive, and one must remember that the pepsin present in the gastric filtrate has been already partly used in the proteid digestion of the test-meal. Sahli's desmoid-proteid digestive test will be considered under the absorption tests.

Tests for Rennin and Rennin Zymogen.—To 10 or 15 c.c. of neutral milk add 5 c.c. of gastric filtrate, and place the mixture in a thermostat or in any warm place. In 10 to 15 minutes coagulation begins. This is merely the familiar "junket making." Free HCl is not necessary for its performance.

Quantitative Tests.—For the differentiation of many of the disorders of gastric secretion an estimation of the amount of acids and acid combinations is often necessary. Free HCl is the only free acid regularly estimated. The estimation of the total acidity of the gastric contents, which is made up of free HCl, traces perhaps of other acids (lactic acid), and combinations of HCl and lactic acid (if present) with the proteids of the admin-

istered meal, is the next important step. Estimation of these combined products are also made.

Quantitative Estimation of the Amount of Free HCl.—The amount not used in the process of digestion at the time of the test-meal's removal. The general principle of all the tests for total acidity and free acids is the same, namely, to add to the filtrate a standard alkaline solution until the acid contents are neutralized. To aid in determining neutralization various coloring agents, some of which have been already described in the qualitative tests, are added to the gastric filtrate. These coloring agents have the peculiarity of losing or even changing their color when the filtrate becomes neutral or faintly alkaline in reaction from the added alkali. The solution in general use is the "one-tenth normal" sodium hydrate. This one-tenth normal solution, written $\frac{N}{10}$ NaOH, is preferred on account of its dilution, which, when dealing with such small amounts and percentages as are found in the stomach contents, is very necessary. One c.c. of this $\frac{N}{10}$ NaOH corresponds to or exactly neutralizes .00365 gramme of free HCl.

Technic.—Dilute 10 c.c. of the gastric filtrate with 50 c.c. of sterile water and place in a glass beaker. Drop from a graduated burette $\frac{N}{10}$ NaOH until the so-called indicators or coloring agents show the characteristic changes indicative of complete neutralization. The number of c.c. of $\frac{N}{10}$ NaOH used, multiplied by the free HCl equivalent of 1 c.c. $\frac{N}{10}$ NaOH (.00365 gramme), gives the amount of HCl in the 10 c.c. of gastric filtrate, and one readily calculates the percentage amount therefrom, normally about .1825 gramme per 100 c.c.

At present it is more common to express the results in direct figures or per cent., indicating merely how many c.c. of $\frac{N}{10}$ NaOH are necessary to neutralize 100 c.c. of the gastric filtrate (*i.e.*, its contained free HCl), as, for instance, if 10 c.c. of gastric filtrate (containing free HCl) are neutralized by 5 c.c. of $\frac{N}{10}$ NaOH, the percentage of free HCl is said to be 50.

The most convenient indicator for free HCl is dimethylamidoazobenzol. In contact with free HCl in the filtrate a bright red color is shown. Neutralization by $\frac{N}{10}$ NaOH turns the red color to a turbid yellow. The beaker containing the filtrate and indicator should be well stirred or shaken while adding the alkaline solution from the burette.

Equally satisfactory for quantitative estimation is the phloroglucin-vanillin test. Ten c.c. of the filtrate are placed in a beaker, the $\frac{N}{10}$ NaOH is added slowly, and after every 10 to 15 drops one takes a drop of the filtrate and tests it for free HCl on a porcelain plate with the phloroglucin-vanillin. The non-appearance finally of any trace of the carmine-red color indicates the complete neutralization of the free HCl in the filtrate.

The Saahli Test.—The reagent is a mixture of equal parts of a 48 per cent. solution of potassium iodide and an 8 per cent. solution of iodate. Free hydrochloric acid added to this reagent produces free iodine.

Technic.—One c.c. of strained gastric contents is diluted with 15 c.c. of water, and 2 c.c. of the reagent added. Set the mixture aside for several minutes, then titrate with $\frac{N}{100}$ sodium thiosulphate until but a faint yellow color remains. Add a few drops of a 1 per cent. solution of soluble starch and titrate until the blue color disappears. $\frac{N}{100}$ thiosulphate is equivalent to $\frac{N}{100}$ alkali. Therefore the number of c.c. used represents the num-

ber of $\frac{N}{100}$ necessary to neutralize the free hydrochloric acid in 1 c.c. of gastric contents. This is multiplied by 10 as in former estimates.

Estimation of Total Acidity.—The same methods are used with merely different indicators: either phenolphthalein or rosolic acid. Two or three drops of a 1 per cent. alcoholic solution of the former give to the gastric filtrate a turbid appearance. Upon the gradual addition of the $\frac{N}{10}$ NaOH there appears a red-purple color where the drop strikes, quickly disappearing at first but becoming more and more persistent until shaking the filtrate no longer causes the color to disappear. A good rule to follow in this test is to consider the reaction complete when the color will remain for 40 or 60 seconds.

Upon the addition of 2 or 3 drops of a concentrated solution of rosolic acid to 10 c.c. of the filtrate the color is changed to light brown. Neutralization is shown by the appearance of a rosy red color.

Since the estimation of the total acidity requires the greater amount of alkaline solution, it is possible to make both tests in one beaker containing 10 c.c. of filtrate. Using dimethylamidoazobenzol as an indicator one can find first the amount of $\frac{N}{10}$ NaOH necessary to neutralize the free HCl present. By adding phenolphthalein or rosolic acid to the now light-yellow mixture the determination of the total acidity can be made; the amount of $\frac{N}{10}$ NaOH dropped in after using the last indicator being merely added to the amount recorded in the estimation of the free HCl.

Frequently it is of interest to estimate how much of the secreted HCl has combined with the proteid of the meal forming the so-called combined HCl. Many cases showing no free HCl on tests will show that there has been free HCl secreted in the stomach as evidenced by the existence of its combined products.

The simplest tests require the finding of the total acidity in the beginning.

The total acidity represents free acid. acid combined with proteids, and acid salts (acid phosphate).

Alizarin as an indicator reacts acid to free acid and acid salts, but not to combinations of acids and proteids; hence the difference between two tests, the amount of $\frac{N}{10}$ NaOH used in one with phenolphthalein as an indicator, the other with alizarin, must represent the acids in combination. The reaction is complete when the yellow of the indicator turns to a distinct violet.

To summarize these tests with an example, the following normal figures may be used:

1. 10 c.c. of gastric filtrate with phenolphthalein as an indicator for neutralization require 4 c.c. $\frac{N}{10}$ NaOH: 100 c.c. would require 40 c.c. Free acids, acids in combination, acid salts = total acidity 40.

2. 10 c.c. of gastric filtrate with alizarin as indicator (free acids, acid salts) require 3 c.c. $\frac{N}{10}$ NaOH: for neutralization 100 c.c. would require 30 c.c. Total acidity 40 — 30 = 10. Combined acids 10.

3. 10 c.c. of gastric juice with dimethylamidoazobenzol as indicator (free hydrochloric acid only) require 2.5 c.c $\frac{N}{10}$ NaOH for neutralization. Free HCl therefore = 25, in terms of 100.

A much more reliable method of estimating combined HCl is that of

Cohnheim and Krieger. Calcium phosphotungstate separates HCl from its combination with albumin and albumoses, the calcium uniting with the HCl forming neutral calcium chloride. In the process a reduction of the total acidity takes place, corresponding to the amount of combined HCl, which has been changed to the neutral calcium chloride. The difference between titrations before and after the calcium phosphotungstate reaction must represent the amount of acid in proteid combination. The detail is more troublesome than the simple alizarin process, but gives far more accurate results than can be expected where two separate color changes are required.

Four per cent. phosphotungstic acid is neutralized by gently boiling with calcium carbonate. Calcium phosphotungstate is formed; the solution is filtered, tested for neutrality, and can be kept for any length of time. 30 c.c. of this calcium phosphotungstate are added to 10 c.c. of gastric juice. A heavy precipitate of proteid phosphotungstate results while the newly formed neutral calcium chloride remains in solution. This mixture is now filtered, the precipitate remaining on the filter paper being well washed by pouring on it distilled water (two or three separate additions of 5 or 10 c.c.) and adding the wash water to the original filtrate.

Using rosolic acid as an indicator the total acidity of 10 c.c. of gastric juice is first estimated, then the same test is repeated with the material obtained after the phosphotungstate reaction, usually about 50 c.c. of clear fluid. As an illustration:

1. Total acidity of 10 c.c. gastric juice, rosolic acid as indicator = 50.

2. Total acidity of mixture (10 c.c. gastric juice + 30 c.c. calcium phosphotungstate + wash water), rosolic acid as indicator = 35. 50 — 35 = 15, difference due to conversion of HCl combined with proteids into neutral calcium chloride. Combined HCl therefore = 15.

Gastric juices in which the free HCl is absent are often examined for free HCl deficiency. This is necessary if the calcium phosphotungstate method of estimating combined acids is used. The process is simple and similar to the above tests. To 10 c.c. of filtrate, dimethylamidoazobenzol is added. With absence of HCl there is of course no reaction. $\frac{N}{10}$ HCl is now added until a reaction for free HCl takes place. If for instance 1 c.c. $\frac{N}{10}$ HCl must be added, the equation is 10 c.c. gastric filtrate, with dimethylamidoazobenzol as indicator, required 1 c.c. $\frac{N}{10}$ HCl to produce a reaction of HCl. In terms of 100, HCl deficit = 10.

Quantitative tests for lactic acid are not necessary. The chlorides as a general rule are not tested. Since, however, their increase in gastric carcinoma has been claimed, a quantitative estimation is at times called for. The procedure is lengthy, and for its methods the reader is referred to works on chemistry.

Test of Gastric Absorption. — The absorptive power of the stomach may be estimated by the administration, when the organ is empty, of a gelatin capsule containing 0.2 Gm. of potassium iodide. The saliva and urine are tested at intervals of several minutes by the addition of a small quantity of starch meal or a bit of starch paper and HCl. A positive reaction is shown by the familiar blue color which normally should appear

in the saliva in six to fifteen minutes and in the urine in about fifteen minutes. This test is of no great value.

Sahli's Desmoid Test.—More as a test of peptic activity than of gastric absorption, this deserves mention and description. Recognizing that peptic digestion as shown in the test-tube represents by no means the conditions inside the stomach, many investigators have endeavored to invent some capsule which would open and give out its absorbable contents as a result of gastric digestion alone. Great trouble was experienced for two reasons. First, osmosis between the contents of the capsule and gastric juice took place through the animal membranes (proteid substances), and, secondly, many of the substances used were disintegrated by the muscular action of the stomach. Sahli's invention consisted in enclosing absorbable substances (iodoform and methylene blue) in a small piece of rubber dam, tying them in with a strand of raw catgut. Osmosis cannot take place through the rubber; raw catgut can only be dissolved by the gastric contents, resisting absolutely the pancreatic ferments. The appearance of iodine in the saliva and methylene blue in the urine is held to indicate that the raw catgut has been digested by the gastric juice and set the contents of the "pill" free; hence the main value of the test is the proof of the digestion of proteid and peptic activity. The details of the desmoid test are as follows:

Iodoform .1 gramme and methylene blue .05 gramme are enclosed in a square of rubber dam 2 × 2 cm. The rubber is stretched tightly to make a small pill and its loose ends tied with catgut which has been previously softened in water. All free hanging edges of rubber are trimmed off. The pill properly formed should sink in water and should show no diffusion of methylene blue when placed therein. Well made and tested in this way a pill given during a full general meal, preferably at mid-day, should sink to the bottom of the stomach and will not be carried off until the end of digestion. In from 5 to 7 hours the first blue tingeing of the urine from methylene blue takes place. Iodine can be determined in the saliva or urine by shaking a small quantity of the respective fluids with a few c.c. of chloroform and adding pure colorless nitric acid, a reddening of the chloroform being the indicator of the presence of iodine.

Peptic activity is most conveniently determined by means of the Mett glass tubes filled with coagulated albumin and submitted to the action of the gastric juice for a definite length of time. The length of the column of albumin digested at each end of the tube is determined accurately with a low power microscope and measured with a millimeter scale. The figure adopted is the mean of both ends.

Since the digestibility of egg albumin varies greatly the method of Nirenstein and Schiff is recommended (Hawk). The tubes are prepared by Christiansen's method as follows: The whites of several eggs are strained through cheesecloth. Glass tubing 8–10 inches in length and 1–2 mm. in diameter is sucked full of egg albumin and laid in a horizontal position. A large evaporating dish is filled with water which is heated to the boiling point and allowed to cool to exactly 85° C. The tubes are placed in the water at 85° C. and allowed to remain there until cool. These tubes contain soft boiled material which is more readily digested than if hard boiled.

The ends are sealed by dipping in melted paraffin and when ready for use marked with a file and broken into pieces of appropriate lengths—three-fourths inch. Christiansen gives the following method for controlling their digestibility: 1 c.c. of gastric juice is put into a small Erlenmeyer flask and 15 c.c. of $\frac{N}{20}$ HCl added. The flask is stoppered and placed in an incubator for twenty-four hours at 37° C. At the end of that time the tubes are removed and the amount of digestion calculated at both ends of the tube, and the mean of the tube readings adopted.

The peptic power is expressed by the square of the number of mm. digested. A reading of 2 mm. gives a digesting power of 4, or in the undiluted juice 4 times 16 or 64.

Tests of the Motor Power of the Stomach.—Lavage.—A satisfactory test for gastric motility consists in the administration of a Riegel test-meal and the washing out of the stomach at the end of seven hours, when under normal conditions the organ will be found to have emptied itself. After an Ewald test-meal traces of food should have disappeared at the end of two hours. No remnants of an ordinary supper should be found upon washing out the stomach the following morning after rising at the usual hour. In atonic conditions and dilatation remnants of partially digested food may be washed out not only at the end of these periods but in extreme cases even at the end of two or three days.

The Salol Test.—Less reliable is the administration of one gramme of salol in gelatin capsules directly after an ordinary meal. The urine is voided at subsequent intervals of half an hour, one, two, three, and twenty-seven hours, and the respective discharges preserved for examination in separate vials. Each portion is then separately tested for the presence of salicyluric acid by the addition of a small quantity of a solution of ferric chloride, which develops in the presence of the acid a violet color. The presence of salicyluric acid in the urine is the sign of the decomposition of the salol into phenol and salicylic acid, and, as this takes place only in an alkaline medium, it is the indication that the salol has passed from the stomach into the intestine, which with normal gastric motility takes place in about one hour. A retarded reaction indicates impairment of motility, a delay of twenty-four hours is suggestive of pyloric obstruction. This test is not accurate, as it is impossible to determine in different individuals the relative time consumed by the chemical changes in the intestine and the elimination by the kidneys. Moreover, the salol may go out of the stomach not with the first portion of the food but with the last. Normally all of the salicyluric acid should have been eliminated within twenty-seven hours.

Occult blood may be demonstrated by the tests described elsewhere in the examination for blood. Wagner's method is probably the most satisfactory. Smears are made of the gastric material to which is added in equal parts a concentrated solution of benzidine in glacial acetic acid and peroxide of hydrogen solution. If blood is present a bluish-green color will soon appear.

(c) **Microscopical Examination of Gastric Contents.**—With the ordinary Ewald test-meal little is to be learned by microscopical examination. Starch granules, a few epithelial cells, and bacteria are usually seen. If there has been much trouble in passing the tube a few blood-cells may be

found. With a mixed meal or in vomited material starch, potato starch, fat droplets, and meat fibres are readily recognized. Many and larger bacteria are seen, a few leucocytes are commonly met with, and in sediments deposited after standing, many large granular mononuclear cells. In cases of acute gastritis considerable blood and pus may be found among the stringy, transparent strands of mucus. In gastric ulcer, blood in the test-meal or vomitus is common. It may be recognizable if the hemorrhage has been recent. The hyperacid condition of the gastric juice in these cases, however, destroys the blood-cells rapidly and chemical tests for the blood are necessary. Tissue cells from the ulcerating area are often found. In gastric cancer with lessened acidity blood-cells are less quickly destroyed, but as a general rule the digestive juices rapidly alter the separate cells. Small clots which have partially resisted the gastric juice form the sediment in the characteristic "coffee-grounds" vomitus of cancer of the stomach. Occasionally small masses showing distinct adenocarcinomatous arrangement may be found and are conclusive proof of the existence of cancer. Small masses of tumor visible macroscopically are occasionally seen in the vomit or washings from a carcinomatous stomach.

Of the bacilli present a majority are small, more or less motile, probably introduced with food. A few extra large organisms of the hay bacillus group are always to be found. One should be careful not to consider these large regular organisms as the form described by Oppler and Boas. The latter are large, irregular club-shaped and vacuolated bacilli, possibly the degenerate forms of the so-called gas bacillus, or of a special lactic acid forming bacillus. They are most commonly found when lactic acid is present, and under this condition have been considered as suggestive of cancer.

Einhorn's Bead-test of Digestive Activity.—Six small glass beads are connected with a silk thread; to each bead is tied some particular sort of food. Raw catgut and a soft long-bone of a pickled herring are the two substances used to test gastric digestion; raw meat, raw thymus gland, mutton fat, and a cube of cooked potato test the intestinal digestive power. The beads and thread can be placed together in a gelatin capsule and swallowed. Normally the beads should appear in the stool in one or two days; their elimination earlier than this indicates accelerated motility of the intestine; their appearance in the stool later than two days after administration is held to indicate retardation of the fecal excretion. All the beads should be empty, though traces of fat, thymus, and fish-bone may be left undigested. Excretion of the catgut and fish-bone undigested would indicate impaired gastric digestion. Excretion of undigested meat, thymus, or fat indicates deficient intestinal digestion. The silk thread is of course merely to facilitate the finding of the beads.

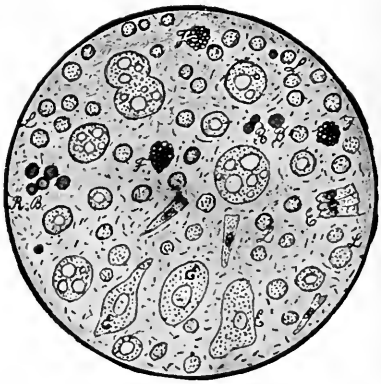
The Fractional Method of Gastric Analysis.[1]

The Fractional Tube.—The older methods of gastric analysis were repeatedly found so inaccurate in Hawk's Laboratory that a new procedure was developed by Rehfuss—the so-called "Fractional Method." The accompanying illustration shows the impossibility of interpreting gastric work

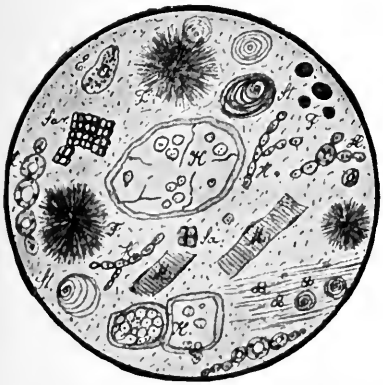
[1] Contributed by Martin E. Rehfuss, M.D., collaborator.

Plate IIA.

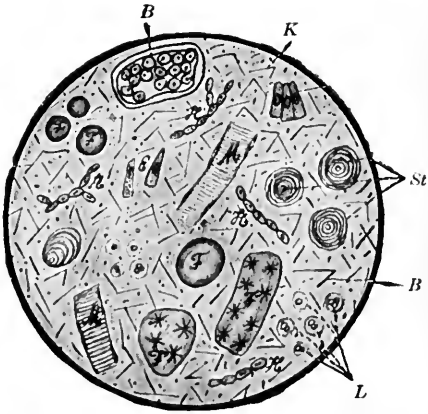
K, free nuclei; *Sp*, spirals; *Sch*, mucus; *H*, yeast-cells; *E*, epithelium; *AE*, alveolar epithelium.—Cohnheim.



E, epithelium; *L*, leucocytes; *RB*, red blood-cells; *F*, fat-cells.—Cohnheim.



St, starch-cells; *H*, yeast-cells; *Sa*, sarcinæ; *M*, muscle-fibres; *F*, fat-balls and droplets; K, potato-starch cells.—Cohnheim.



H, yeast-cells; *M*, muscle-fibres; *L*, leucocytes with shrunken nuclei; *B*, Oppler-Boas bacilli; *St*, starch-cells; *F*, fat; *E*, epithelium; *K*, potato-starch-cells with yeast-cells.—Cohnheim.

from a single examination, the one-hour investigation being susceptible to the various interpretations shown in the drawing, each of them being of different significance.

A special tube has been devised, the "Rehfuss Stomach Tube," which can be left in the stomach for long intervals without inconvenience to the patient. Rehfuss has left the tube in the stomach for days without bad effect. The tube is of small bore (No. 10–12 French) 100 cm. in length to permit both gastric and duodenal work, glass moulded and fitted with a metal tip of sufficient weight to gravitate to the bottom of the stomach. The slots are the same size as the tubing in order that anything which passes through the slot will pass through the tube. An aspirating syringe makes the apparatus complete. The tube is introduced into the stomach

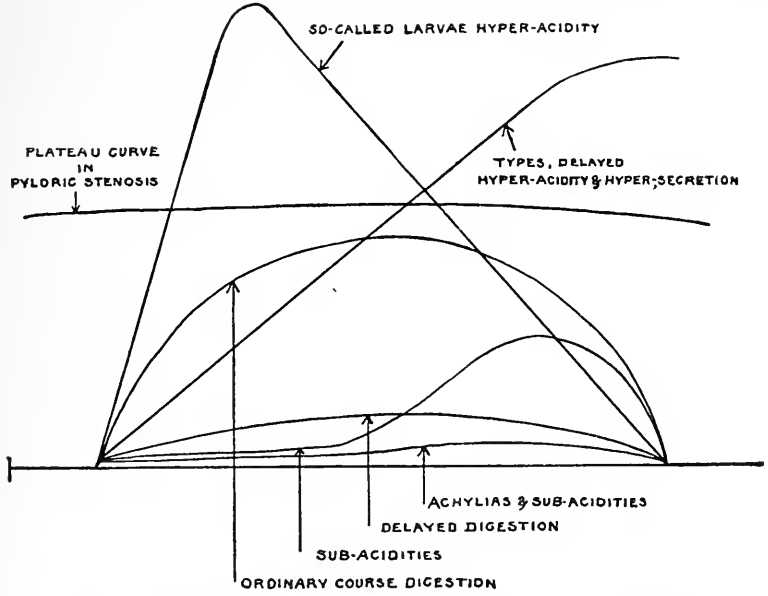


FIG. 84.—Possibilities in the evolution of the secretory curve of digestion.

by swallowing and not by propulsion which is used in introducing the older type of tube. With this instrument it is possible to follow every phase in gastric digestion. Rehfuss has studied the changes occurring after the ingestion of all varieties of food.

Method of Introduction.—The tip of the tube is lubricated with vaseline, glycerin, or lubricating jelly. False teeth or other movable objects are removed from the mouth. The physician grasps the tip between the thumb and forefinger and passes it along the back of the tongue until it engages in the pharynx. The patient is encouraged to swallow—this carries the tube into the stomach. Any difficulty is overcome by deep breathing or by the drinking of water. Many patients are able to swallow the tube without any help whatsoever. A supersensitive pharynx may be painted or sprayed with a 2 or 4 per cent. solution of cocaine hydrochloride. One may make several applications to the root of the tongue and to the epiglottis. This is exceptional and not a routine procedure.

There are certain points which are of practical value. The tube gradually enters the stomach. That it is still in the *œsophagus* is shown by the following points: (1) aspiration removes a small sample of saliva or œsophageal mucus, or (2) it discloses a block, no material being aspirated owing to the closure of the slots by the œsophageal walls; (3) injection of fluid occurs readily, but cannot be reaspirated because it has entered the stomach.

When the tube enters the "air chamber" it is recognized by the fact that: (1) aspiration brings back air, (2) injection is easily performed but no liquid can be reaspirated.

The gastric secretion is obtained and readily recognized when the tube enters the fundus. Inability to recover material is due to one or more of the following factors: (1) an unclean tube. It should always be tested before using; (2) the tube is in the œsophagus, cardia or air chamber; (3) the tube is blocked by material too coarse to pass through it; (4) too much tubing has been passed and the tip may be in the pylorus; (5) occasionally hyperactive peristalsis may tie a knot in the tube.

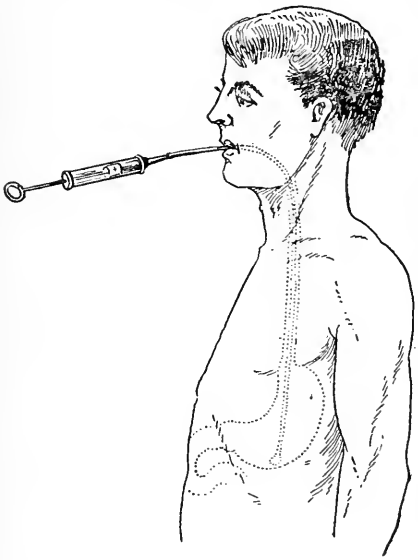


FIG. 85.—Stomach tube in position.

The end point of gastric digestion or the exact point of gastric evacuation is determined by means of the following points: (1) No more food can be aspirated, (2) by a change in the appearance of the samples and the addition of a little pyloric mucus, (3) the injection of water into the stomach and its reaspiration demonstrates the absence of all food, (4) when water is injected auscultation will reveal a hissing instead of a gurgling sound. Ordinarily about 55–60 cm. of tubing is passed, depending upon the position and size of the stomach.

Gastric Lavage.—Is performed readily with this tube. The correct time for lavage with the fractional tube is when the stomach is empty. Various lavage liquids can be introduced into the stomach and reaspirated; if normal salt solution is used the material removed can be centrifugalized, stained and cytological studies made. This often reveals interesting data in gastric conditions.

Inflation and Auscultation.—Can be performed. The stomach is easily and completely inflated with a bulb, and the outlines of the organ determined by palpation or percussion. It is interesting to combine auscultatory study with inflation. When the viscus is full of material—especially hypersecretion or liquid—a number of small gurgling râles are heard; as the organ is evacuated, the râles become discontinuous and "sticky." Finally, when the organ is completely evacuated, after a few initial sticky râles, the rush of air is distinctly heard and fades out as the stethoscope is placed over areas other than that of the stomach.

The Syphon Method (Fig. 86).—The end of the tube is allowed to

drain over the edge of the bed and the gastric specimens accumulated in tubes. Hyperseeretion can be studied in this way and affords considerable

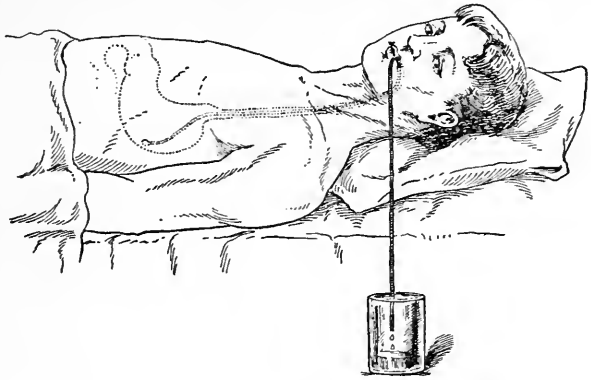


FIG. 86.—Syphon method of obtaining gastric secretion.

information of value. Furthermore, the determination of occult blood obviates the possibility of trauma in the collection of the sample.

Medicaments, such as silver, iodides and various disinfectants, can be made and controlled with the fractional tube. The tube may be introduced into the stomach, or the duodenum, or both, and fluids with or without medication administered by the drop method similar to the Murphy drip used in intestinal work.

Gastric Intubation.—The direct study of gastric work has undergone considerable alteration. It is possible to study every phase of gastric digestion and to demonstrate the alterations in disease. A single examination at a definite time after the administration of a test meal was formerly thought sufficient to determine gastric efficiency. The introduction of the fractional gastric tube has totally altered this conception. It is realized that there are two periods of gastric activity, one the digestive period in response to the stimulus of ingested food (Fig. 87), the other the interdigestive or rest period during which the gastric walls are more or less approximated by peristole while peristalsis practically ceases. Tonal or hunger contractions at intervals take their place. Gastric digestion consists of an evolution of changes and a single examination of the meal gives no evidence of what precedes or follows that point in digestion. This has been determined by studies made upon a large number of normal individuals. These changes are altered in disease not only in the character of

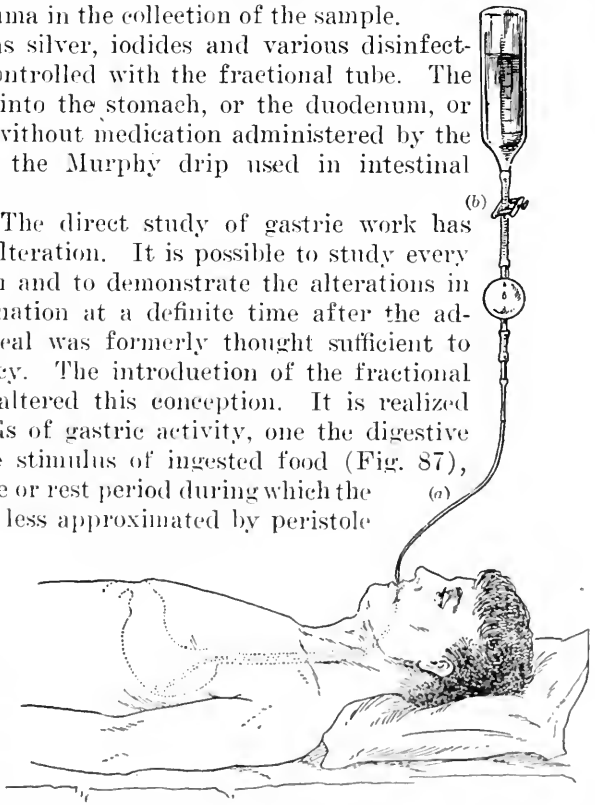


FIG. 86a.—Medicaments applied to stomach. *a*, Rehfuss tube; *b*, Murphy drip.

their evolution but by the addition of products characteristic of disease, *viz.*, mucus, pus, blood, bacterial products and the specific products of the diseased process. Gastric intubation will give not alone the type of gastric digestion but also the evidence of disease—made possible by a study of the products enumerated above.

The residuum is the material obtained from the stomach during the interdigestive (rest) period. It is usually obtained in the morning. It normally averages 30–50 c.c., is thin, opalescent, contains bile in over one-half the cases, has an average total acidity of 30, and an average free acidity of 18–20. It may contain gastric mucus equally distributed throughout the specimen as differentiated from swallowed mucus, which is aerated and floats on the specimen. It should contain no pus, blood or macroscopic food. The residuum enables one to differentiate between swallowed material and exudation from the gastric wall. It demonstrates the presence of retention which may be intermittent in spasm, or permanent in gastric disease—intragastric from indurated ulcer, neoplasm near the pylorus, a hypertrophic pylorus, or weakness of the gastric wall as found in the atonies

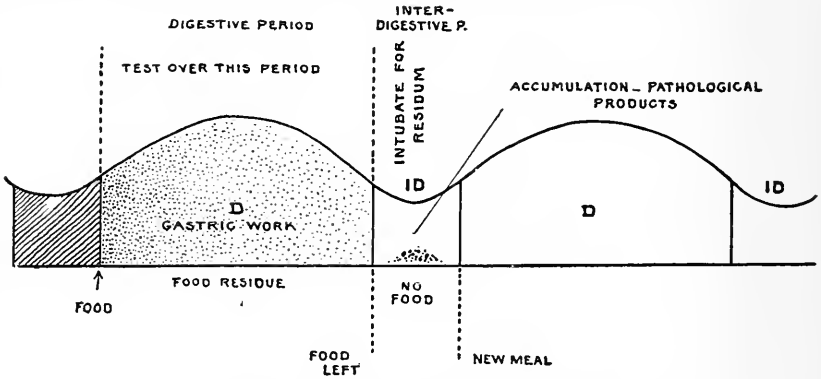
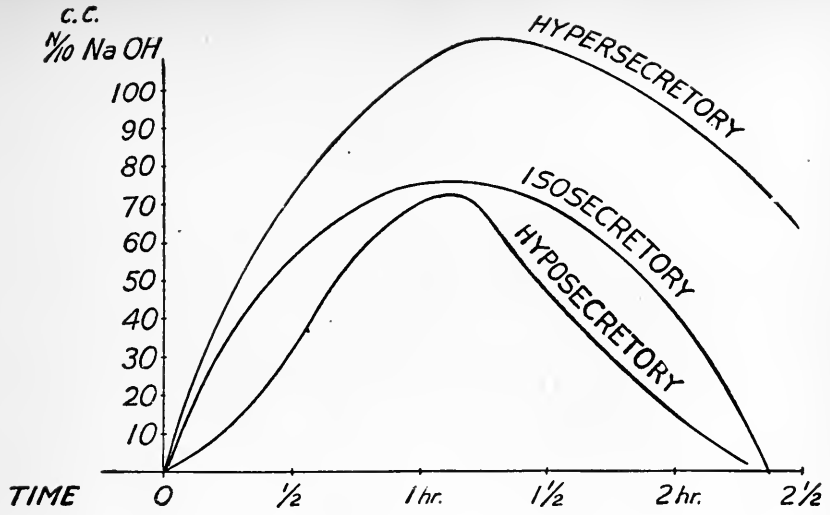


FIG. 87.—Digestive and interdigestive periods.

and myasthenias in the various forms of dilatation, or extragastric from adhesions and tumors of the gall-bladder and pancreas, etc.

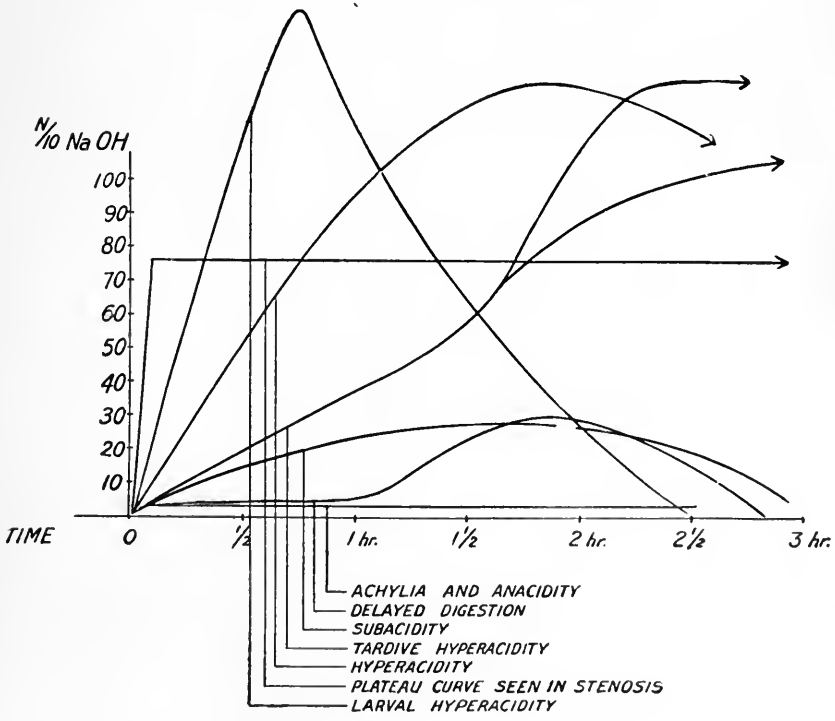
Fractional Analysis.—Remove by gentle aspiration 5–10 c.c. of gastric material after the administration of the test meal. Repeat at fifteen minute intervals until the end point in gastric digestion has been reached. The interval may be lengthened to twenty or thirty minutes if digestion is delayed. Collect the specimens in a series of labelled tubes and examine each specimen separately. The material is strained through cheesecloth and the filtrate examined chemically by the same methods used when the single one-hour gastric specimen is obtained, excepting 1 c.c. of filtrate is used instead of 10 and $\frac{N}{100}$ NaOH is used instead of $\frac{N}{10}$ NaOH. In calculating the percentage this fact must be kept in mind, and the reading multiplied by 10 to determine the number of c.c. of $\frac{N}{10}$ NaOH necessary to neutralize 100 c.c. of gastric material.

Duodenal Intubation. — This method is important and is rapidly becoming a routine procedure for the study of the bile and the pancreatic secretion. The Rehfuss tube or the duodenal tubes of Einhorn and Jutte are employed, about 75 or 80 cm. of the tube being passed as for gastric



NORMAL CURVES

FIG. 88.



PATHOLOGICAL CURVES

FIG. 89.

analysis. Clear broth or bouillon is given. The patient is turned on his right side with the pelvis rotated, the left leg swung over the right and maintained in this position. The pelvis may be elevated by placing the pillow under the hips. Gentle aspiration is performed in three-quarters of an hour and repeated at fifteen minute intervals until bile is obtained. This is readily recognized by its color, general appearance and viscidity. When bile is secured gentle aspiration may be used to secure other specimens. The syphon method, however, is the most satisfactory. The same chemical, microscopical and bacteriological examinations are made as in the examination of other body fluids.

Gastro-Duodenal Intubation.—Gastro-duodenal intubation for simultaneous examinations of the stomach and duodenum can be performed by the Rehfuss double gastro-duodenal tube or by the Palefski tube, or more easily by primary duodenal intubation, followed by gastric intubation after bile has been secured from the duodenal tube. Samples can be secured in series as already described.

The gastric curve in health is shown in the accompanying illustration. There is no curve which can be considered as ideal, inasmuch as Rehfuss' studies of normal individuals showed that they reacted differently to the same stimulus, but in general all follow the same trend. The response is exaggerated in one—the hypersecretory type—and in another somewhat sluggish—the hyposecretory type. The evacuation time is generally between two and two and one-half hours with an Ewald meal. Premature evacuation occurs in achylia, gastric and the subacidities, in certain forms of scirrhous carcinoma, in a small proportion of duodenal ulcers, and of nervous hyperperistalsis. Delayed evacuation is seen in a multitude of conditions with a slight delay in the atonies, and in some forms of ulcer pronounced delays are seen in pyloric obstruction, in indurated ulcer or neoplasm at the pylorus.

The secretory variations are many (Figs. 88 and 89). We may have an absolutely flat curve in achylia (without enzymes) and anacidity, or low curves in the subacidities and the delayed responses. We may have premature or larval hyperacidity or the long delayed hyperacidity so frequently associated with post-digestive hypersecretion. Again, we may have a clean digestion associated with secretory variations, or these variations may be associated with all the phenomena of intragastric lesions, pus, blood, mucus, bacteria—neoplasm, syphilis, tuberculosis, and infective gastritis. Lesions in the gall-bladder, appendix and other parts of the abdomen may alter the secretion through the vagus. Cardiac incompensation and portal hypertension incident to hepatic cirrhosis may induce secondary gastritis with mucus and secretory alterations, etc.

EXAMINATION OF THE INTESTINES AND FÆCES.

Only the lower bowel is accessible for direct examination of its interior. Inspection with the aid of proctoscopes, digital examination of the rectum, and inflation of the colonic area enable us to investigate at least a part of the large bowel directly. The remainder as well as the small intestine can only be reckoned with through the abdominal wall by inspection,

palpation, percussion, auscultation, and radioscopy. On the other hand, a careful examination of the fæces will tell us much as to function of the intestines and as to the presence of abnormal conditions.

Inspection as applied to the examination of the intestines may be, as we have said, direct when we are dealing with the large bowel; the proctoscope and the rectal tubes allowing inspection of the mucous membrane practically to the splenic flexure of the colon. The tubes or specula come in varying sizes, usually four in a set, ranging from 4 to 14 inches (14 to 35 cm.) in length and from 1 inch diameter in the short speculum to ½ inch diameter in the longer. They are provided with obturators. Their use is associated with considerable pain, though with persistence and gentleness most patients can go through the performance without an anæsthetic. Warming and oiling the instruments thoroughly will overcome some of the difficulties. After the sphincter muscle of the anus has been stretched and dilated the discomfort lessens. Either the knee-chest or the recumbent posture with the knees elevated may be used. Too much elevation of the lower part of the body will naturally by gravity send the bowel away from the examiner. The electric headlight with reflector facilitates examination. Very little trouble is experienced in straightening out the sigmoid flexure, nor do the valves of the rectum interfere with the progress of the speculum. It is common to find that the bowel dilates perceptibly with air when the speculum is in place, thus materially aiding the examination.

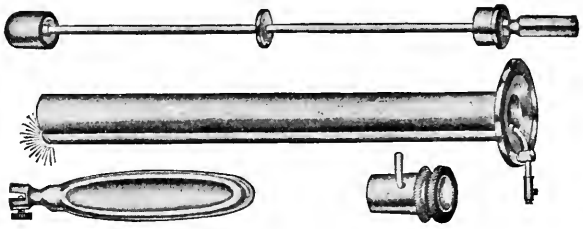


Fig. 90.—Rectoscope.

In the more modern rectoscopes and sigmoidoscopes the distal end is so arranged that air can be forced into the bowel in front of the advancing tube. A glass shield near the distal end allows the observer to look into the bowel and at the same time keeps in the injected air which balloons the bowel for several inches. A small electric light arranged in front of the tube gives a clear view of the mucous membrane.

Inspection with the proctoscope or rectoscope may show us first the presence of scybala, beyond reach of the palpating finger, revealing themselves often as hard, adherent, though detachable, masses of varying size, dark in color, or gray if covered by mucus, and usually easily indented or broken away with a probe, and readily differentiated from polypoid and other growths; second, ulcerating and bleeding points, dilated venules, fistulous communications, thickening and reddening of the mucous membrane of the bowel; general reddening of the whole surface in colitis, showing mucus, glairy or dense and white if the condition of mucous colitis is present, and often in large amounts; third, polypoid growths, malignant growths in the form of local thickening, strictures, foreign bodies, ulcerating tumor masses; fourth, obstructions outside the bowel, tumors of uterus, ovary, etc., preventing insertion of the examining

tubes. It is usually considered permissible and advisable to remove small particles of suspicious growths for microscopical examination.

Examination of the bowel by means of bougies may at times help in diagnosing a stricture, particularly if the feeling of distinct resistance and the sensation of passing an obstruction can be appreciated repeatedly at the same point both during the introduction and withdrawal of the sound.

Palpation as a means of examination in regard to the intestine has but limited direct application, namely, the examination of the rectum by the finger, or if the sphincter can be dilated by two fingers or the whole hand. Examination is always facilitated by a previous good clearing out of the lower bowel. The forefinger of either hand may be used and various positions employed. The examiner, however, reaches a higher point in the bowel if the patient lies on his side with his knees drawn up to a moderate extent. In this posture more freedom is given the introduced finger, and the rotation of the hand and finger in examining the sides and front of the rectum much aided. The knee-chest position is also convenient.

To avoid the unpleasant fecal odor it is advised to fill the space between one's nail and finger with a little moist soap and to follow this by a free greasing of the finger with oil or vaseline. A finger cot can be used.

After inspecting the anus and neighborhood for fissures, fistulæ, hemorrhoids, exuding blood or pus, etc., the finger is gently inserted, overcoming gradually the spasm of the sphincter which always occurs and which must not be taken for a stricture. The tight grip of the muscle on the finger relaxes during the examination and gives considerably more freedom of movement. Examining as a routine the prostate and bladder, or the uterus, one may at once exclude or determine conditions affecting those organs; then, sweeping around to either side one feels for points of tenderness, irregularities on the smooth wall of the bowel, dilated veins, fistulous communications, polypi, etc. The finger is then turned toward the back of the rectum. The position and condition of the coccyx should always be determined during any rectal examination. Various obstetrical, genito-urinary, and gynæcological examinations may be made by way of the rectum. The fact that impacted ureteral calculi can at times be perceived by the examining finger if caught in the bladder wall or in the neighborhood of the pelvic brim must be mentioned. Too little consideration is given to a rectal examination in appendicitis and appendicular complications. An appendix abscess extending downward, though often painless, is frequently associated with an extreme tenderness when touched by the examining finger. This tenderness is usually too localized to be confused with any general abdominal distress, and its high position to the right is of diagnostic significance.

The examining finger has first 1–1½ inches of contracted sphincter area to overcome. A slow inserting movement dilates the muscles without pain, and allows the examiner and patient to appreciate localized tender areas, such as are occasioned by fistulæ, or ulcerations of hemorrhoidal veins. A forcible dilatation would readily, by the pain occasioned, prevent the disclosure of many of these minor conditions. Beyond the sphincters the finger has free play and at times one fails to touch any part of the bowel, the rectum being ballooned by flatus; by crooking the

finger one touches readily the rectal wall. It is at times possible to distinguish emptied fallen coils of small intestines in the pelvis by rectal examination; such a condition may take place when a complete stricture has occurred high up in the small intestine, the empty tract below collapsing and descending.

Indirectly both the small and large bowel can be examined through the abdominal wall by inspection and palpation. Auscultation has but a doubtful bearing on abdominal conditions; even marked intestinal disorders may yield nothing to the most experienced. On the other hand, correct interpretations of simple existing phenomena may give most important results. Inspection is always preferably performed with the patient lying on his back, with his knees either flexed or extended. (See Methods of Physical Diagnosis.)

Examination of the large intestine and the cæcal region is helped by the easily applied method of inflation. Before this procedure a thorough purging is always advisable. The soft rubber rectal tubes, $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter, with two or more lateral openings, and connected with a Davidson syringe or a double or single atomizer bulb, can be inserted to any distance desired. A slow twisting insertion will quickly put the openings above the anus and sphincters. Since the sigmoid flexure is the most commonly dilated part of the bowel, inflation should begin while the tube is entering the flexure, and the first examination directed to this part. Unless previously distended by gas or continued fecal accumulations the sigmoid flexure should not rise easily out of the pelvis during inflation. Usually when a point half way between the groin and the umbilicus is reached distinct discomfort is felt unless relieved by passage of the air upward. In many instances it will be found that the inflating air passes readily beyond the sigmoid and shows its presence in the descending and transverse colon. It is now generally conceded that the ileocæcal valve allows air to pass during the process of inflation and its passage can at times be heard with the stethoscope applied in the right iliac fossa.

To further inflate the colon the rectal tube may be passed upward its whole length; we cannot be sure, however, that it will pass beyond the splenic flexure nor could further passage be expected. Inflation of the transverse and ascending colon and of the cæcum take place quite readily with the tube in this locality. The pain of extreme distention will always warn the operator when to temporarily moderate the air-pressure, which, however, is usually relieved at this stage by the passage of air upward through the ileocæcal valve. Detaching the rectal tube from the inflating apparatus allows the bowel to return to its normal state, by expelling the contained air.

Carefully applied inflation in connection with inspection and palpation may give important results. Idiopathic dilatation of the sigmoid flexure can be readily differentiated from abdominal distention due to other causes, the sigmoid clearly outlining itself as it rises from and descends again into the left iliac fossa, often overlying the rest of the abdominal contents in its sweep upward and to the right. Tumors, malignant or other strictures of the bowel, fecal accumulations may be brought up from the pelvis into sight and touch. The position of the colon and cæcum can

be readily outlined, visibly in thin subjects, by percussion and palpation in those stouter and more muscular, although the examiner can always see that inflation is going on by the puffing up of the various regions. One must know that at both the splenic and hepatic flexure the bowel will be less prominent than elsewhere. The same pathological conditions mentioned in connection with the sigmoid flexure may be shown in the colon. One would naturally expect that complete strictures from any cause would prevent passage of air upward or downward. In such cases the distention and condition (muscular hypertrophy, visible peristalsis) above the stricture may tell us as much or more than inflation from below; and further, in such conditions the diagnosis is rarely in doubt. Incomplete or partial strictures, whose symptoms may be very obscure, are at times clearly brought out by the rapid inflation from below, as a sudden narrowing above the dilated lower part.

Easily recognized is the displacement of the colon, particularly the transverse colon in enteroptosis. The transverse colon may lie below the umbilicus, or even in the pelvis, the common appearance on inflation being a shallow V-shaped protuberance, the arms of the V running up to the liver and spleen. The relation of the bowel to retroperitoneal and other tumors is more easily determined by inflation than by any other means. Inflation of the bowel above the ileocæcal valve doubtless may be of value. Its application has given but uncertain results. Inflation of the bowel as a test for perforation is now universally condemned. Many cases of flatulence supposedly due to gastric distention can be found to be due to distention of the colon. Inflation is a valuable aid in the differential diagnosis of these conditions.

Röntgen-ray Examination of the Intestines.—Large and small solid tumors, thickening and muscular hypertrophy can at times be made out by the fluoroscope or in skiagrams. The data obtained by this method are, however, usually confirmative of facts elicited by the anamnesis and the above described methods. Scybalous masses present no different shadow from that of organic disease. Lining the intestine by continuous doses of bismuth allows the coils to be readily photographed, and under this condition peristalsis can be readily observed by the fluoroscope and the rate of progress of fecal matter observed. More feasible and of distinct value in recognizing displacements of the colon is the injection of suspensions of bismuth in large quantities. Very serviceable photographs can be secured by this method. Localization of small metallic or other solid foreign bodies in the intestines is remarkably facilitated by the X-rays.

So-called "test lavage" is used at times to bring away secretion or material from the large bowel: mucus, blood, ulcerating fragments of new growths. The examination of the sediment of such washings at times gives distinct help. It can only be satisfactorily performed when the large bowel has been previously completely emptied.

It has been suggested that dilatation of the duodenum, with stricture beyond, can be diagnosed by filling the stomach and duodenum with water through a stomach-tube. Dulness and distention toward or in the right hypochondrium and the fact that the fluid may return as does the fluid

from an hour-glass stomach, part at once and the rest a few minutes later, is considered suggestive. An inflation that outlines the stomach and produces an extra prominence in the right hypochondrium would be equally suggestive.

Fæces.—The accurate determination of many points with regard to the fæces is difficult, owing to the wide variations in their composition and to the fact that the establishment of a normal or standard bowel movement requires the continued administration of certain standard diets for several successive days.

Various standard diets are recommended, the simplest being milk, since it contains fat, proteids, and carbohydrates.

1. Milk, 8 oz. every two hours from 8 A.M. to 10 P.M., amounting to 4 pints in the twenty-four hours.

2. That of Schmidt is more complicated, but approaches more nearly a general diet:

7.30 A.M.	Milk, 17½ oz., and 6 biscuits.
9.00 A.M.	Gruel, 1½ oz. oatmeal, 1 egg, 2 biscuits, ½ oz. butter, 7 oz. milk, 10¼ oz. water.
1.00 P.M.	Minced beef, 4½ oz. raw weight, lightly fried in ½ oz. butter, leaving the interior raw, and potato purée—7 oz. mashed potatoes, 7 oz. milk, ½ oz. butter.
4.30 P.M.	Milk, 17½ oz.
7.30 P.M.	Same as at 9 A.M.

3. A "mixed diet" is more liberal and better borne, but the necessary cooking makes the eventual microscopical examination much less satisfactory than either of the preceding:

8.00 A.M.	10 oz. hot water.
9.00 A.M.	3 oz. fresh fish, 4 biscuits, ½ oz. butter, 10 oz. tea, 2 oz. milk.
12.00 M.	10 oz. hot water.
1.00 P.M.	3 oz. mutton, 3 oz. cabbage, 4 biscuits, ½ oz. butter, rice pudding (½ oz. rice in 10 oz. milk).
4.30 P.M.	10 oz. tea, 2 oz. milk, 2 biscuits.
6.00 P.M.	10 oz. hot water.
7.00 P.M.	3 oz. fresh fish, 3 oz. chicken, 3 oz. spinach, rice pudding (as before), 2 biscuits, ½ oz. butter.
10.00 P.M.	10 oz. milk.

4. A meat diet: ¼ lb. finely minced beef every three hours and 10 ounces hot water one hour before meal-time. It contains no carbohydrates.

The first dejecta usually appear in from twenty-four to forty hours after the standard meal has been given. Radioscopic examination of the intestine and the passage of its contents shows that in about seven hours the ileocæcal valve has been reached by part of the residue, which may now remain four hours in the colon, three hours in the sigmoid flexure and rectum before being expelled.

Attempts to describe bowel movements resulting from standard diets have been made.

1. Fæces Resulting from Milk Diet.

Amount.—

Quantity of milk in 24 hrs.	Fæces excreted, average weight in Gm.
4 pints	135.2 Gm.
5 pints	151 Gm.
6 pints	198 Gm.

Color.—Yellow-white, or white tinged with orange.

Consistency.—Not well formed, tending to be lumpy; rolls of fecal matter not homogeneous but composed of lumps welded together, or firm sausage-shaped masses plus soft paste.

Odor.—Not offensive; more like stale cheese than fæces.

If constipation exists, a tendency to isolated scybala of pale color is seen, often firm, hard, and dry enough to rattle in the vessel, and to break up like dry clay, with an earthy odor.

With diarrhœa a milk diet gives fæces resembling Devonshire cream—sticky, but capable of being poured from one vessel to another. Gas bubbles and froth are seen on shaking, and the odor is that of decomposed cheese or putrid proteid.

Caseous flocculi, the evidences of disturbed digestion, are readily recognized as bright white, small, fibrillary-looking, friable masses.

2. Fæces Resulting from the Schmidt Diet.

Amount.—Smaller than that from the milk diet. Average 90 Gm.

Color.—Light brownish-yellow, darker on the outside than inside.

Consistency.—Well formed rolls or sausage-shaped masses, as a rule. These readily break up on drying.

Odor.—Distinctly fecal.

In constipation on a Schmidt diet lumps of fecal matter are massed together, or isolated scybala are seen.

In diarrhœa on this diet the fæces resemble closely those of a patient on a milk diet.

3. Fæces Resulting from a Mixed Diet.

Amount.—Average 102 Gm.

Color.—Nut-brown, olive-green (chlorophyll of vegetables), varies much from day to day.

Consistency and Form.—Usually large, firm, roll or sausage-like motions. On drying break up easily.

Odor.—Fecal.

In constipation the fæces of a mixed diet are usually dark brown or black scybala with pressure facets and mucus in the crevices. They may be of stony hardness and not offensive.

In diarrhœa the motions are dark brown or nearly black, of thick sticky or pasty consistence with small scybala. Soft movements in general from a mixed diet have most offensive odors. An increase of the quantity of milk in mixed diets makes the stools paler and less firm.

4. Meat Diet.

Amount.—Average 54 Gm.

Color.—Dark brown to black.

Consistency and Form.—Firm rolls, 2 to 3 inches in length.

Odor.—Fecal but very offensive.

Variations in consistency and form, in odor, and in color naturally depend on local conditions and the time the fecal material is retained in the large bowel. The amount is important, but several days are required to get the proper average. The formation of scybala, according to these results, may take place in a very few days.

The fæces are composed of:

1. Food remains.
   (a) Indigestible remnants.
   (b) Digestible but not absorbed remains.
2. The remains of the digestive secretion.
3. Products resulting from the digestion of food in the intestinal canal.
4. Formed and unformed products of the intestinal mucosa.
5. Bacteria.
6. Various substances introduced accidentally from without; various concrements, gall-stones, intestinal stones, parasites, cotton, wool, or linen fibres.

The fæces are collected in a bed-pan or any large clean vessel.

In the study of any question of absorption or excretion the rule is to place the patient on one of the standard diets for at least four days before beginning any estimation. The administration of some coloring matter such as charcoal or carmine with the first meals of the standard diet will render easy the recognition of their first dejecta.

The fæces are to be examined macroscopically, microscopically, and chemically.

Fermentation.—Normal firm bowel movements will usually dry without appreciable gas formation, and even semisolid or pultaceous stools ordinarily produce only a small amount. A stool which on standing shows evidence of fermentation by the production of gas bubbles or a distinct frothy layer, or gas bubbles in such abundance as to give a pale appearance to a more or less solid stool, should be considered pathological and examined for fermentable products—carbohydrates.

Excess of neutral fat in the stools can be readily noted. The normal bowel movement leaves no greasy mark upon a vessel containing it. Neutral fat will show itself in the gross examination either as a very pale, white, distinctly greasy bowel movement, or if the stool be liquid the fat may rise to the top, forming the characteristic appearance of melted fat, and on cooling may partially or completely solidify. The soaps in ordinary amounts and the fatty acids are not macroscopically recognizable.

Excess of proteids in the fæces, when in the form of meat, can often be recognized by the appearance of numerous reddish points throughout the bowel movement. One must be certain that other coloring or colored matters have not been ingested. Casein shows itself as the familiar white flocculi, easily disintegrated, much denser white than mucus. Undissolved connective tissue has the appearance of fine cotton-wool fibres and can be removed for further examination. Other substances to be considered in the gross examination are mucus, blood, pus, foreign bodies, and parasites. Small amounts of mucus are always present, but require search to demonstrate their presence. A constipated stool often shows flakes of dense white mucus in the interstices of the firm masses, or mucus may follow the movement.

Brownish, gelatinous-looking mucus, colored by the bile pigments, usually comes from the small intestine; colorless mucus and that appearing as denser, whiter masses and flakes, from the colon. Tubular masses from the large intestine, sometimes many centimetres in length, are seen

in membranous colitis. Floating or softening these masses or strands in water will usually determine their character. Unformed mucus, particularly in liquid stools, sometimes on standing accumulates in masses as large as a hen's egg.

Fresh blood can be easily recognized. Unless quickly voided, blood in the intestines becomes black and small amounts do not show in the stools. Large amounts appear as "tarry stools"—large black masses, clots too large to be broken up or absorbed.

Pus is usually quickly disintegrated. Fresh pus which retains its appearance is practically always from the sigmoid or rectum.

Parasites are described in another section. Many food remains are detected at a glance: fruit stones and seeds, skin of fruit, vegetables, food pulp of oranges, grape fruit, lemons, large masses of connective tissue, bones, etc.

For the more careful examination various simple plans are recommended. In examining the whole quantity of fæces an ordinary fine sieve on which running water can play enables one to collect the larger foreign bodies and solid material; or the fæces are placed in a large vessel with water and thoroughly broken up. Mucus, woody fibres, smaller seeds, and bacteria float and can be removed by pouring off after settling. By repeating the process several times a residue of solid matter, deodorized and decolorized, is obtained. Gall-stones, pancreatic calculi, muscle fibres, connective tissue, casein, parasites, are easily looked for in this way.

Spreading the fæces on a glass plate with a dark background facilitates the examination. Pieces of connective tissue, muscle fibres, casein, foreign bodies, or anything differing from the homogeneous fecal matter may be readily found in this way.

Microscopical Examination.—Mixed Diet.—A small piece of fecal matter can be taken from the stool after it has been mixed in a mortar or a vessel, or several loopsful of a liquid stool can be smeared on a slide. A cover-glass is preferable if high power is used. For a low-power examination a glass 3 or 4 inches square on which a comparatively large amount of fæces has been thinly spread, can be placed on the stage of the microscope. A large area can be quickly gone over in this way.

Masses of mucus, blood, or pus, meat fibres, etc., should be picked off for separate examination before mixing the fecal material. Schmidt recommends taking three separate specimens of softened fæces. No. 1 is examined direct. In it we can note much fibre, colorless soaps, neutral fat if present, small and large yellow salts of calcium. No. 2 is stirred with a small drop of 30 per cent. acetic acid heated for a moment until it begins to boil, then covered with a cover-glass. After cooling, small flakes of fatty acids appear. The soap flakes and calcium salts will have disappeared. No. 3 is rubbed up with a drop of Lugol's solution. Under the microscope unaltered starch will assume a violet color.

Since 85 per cent. or more of the food is digested and absorbed, and since of the remainder a portion is in the shape of products—albumoses, fatty acid, soaps, dextrin, etc., little unaltered food is present in the specimen. Easily recognized are the bacteria which make up practically one-third of the dry substance of the stool. Acid-fast bacilli may be tubercle

or smegma bacilli. Leptothrix threads are easily recognized. Epithelial cells in considerable numbers are always present. They are usually of the smaller round type, and show evidences of digestion or disintegration. No deduction can be drawn from their number or form as to conditions in the bowel. Squamous epithelia from the mouth or from the food are occasionally seen. Structureless or faintly striated mucus in small amounts, bile-stained if from parts high up, pale if from lower down, may, by the number of leucocytes or epithelial cells entangled in it, give evidence of catarrhal conditions of the bowels. Mucus is less dense, less sharp in outline than connective tissue; acetic acid causes it to show faint striations. A few leucocytes are always present.

Food Remains. — Undigestible remnants of any kind may appear. Many of them are recognizable macroscopically. The framework of vegetables gives most varied pictures. Many of the structures suggest parasites and have frequently been mistaken for them. Remnants of undigested starch may be suspected by their pallid color and their cellular envelope. It is well to stain suspicious specimens with iodine and look for the blue stained masses; to judge whether starch is being excreted in excess is not easy with the microscope; the fermentation test is the more accurate method.

Two or three small meat fibres in a field, showing very dim or no striation, and with no remnants of nuclei, may be considered normal in patients on a mixed diet. Retention of the striation, persistence of the nuclei in good condition, and presence of meat fibres in numbers suggest disturbance of intestinal digestion, particularly that part related to the pancreas. It is not likely that anacidity of the gastric juice will show the same condition. Specimens from fæces of patients with pancreatic derangements may show meat fibres in such numbers that counting them in one field may be difficult or impossible. Some cases show excess of meat fibres in the stools if over 60 grammes of meat are taken per day.

Schmidt's Nucleus Test for Pancreatic Disease or Impairment of Pancreatic Function.—The disintegration or non-disintegration of the meat fibre nuclei in the centre of small balls of meat of standard size—$\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter—kept together by non-digestible netting and given in the food, cannot be said to be positive enough for any certain deductions to be made. We can only say that if all the nuclei, even those on the outside of the balls, are found unaffected by digestion, pancreatic insufficiency is suggested. Connective tissue and elastic tissue are constantly present on a mixed diet, though in very small amounts. They are readily recognized by their dense and fibrillated appearance. Gastric juice readily digests connective tissue, and its persistent presence in large quantity must be taken as pointing to impaired gastric digestion. A few fat drops may be found on a mixed or meat diet, but more than eight to ten fat drops in a single field should attract attention. This neutral fat is easily seen as yellowish, oily looking drops of varying size and shape. Constant presence of the flakes of the "higher melting point" fat, and the flaky needle-like crystals of the fatty acids, or of the flake or disk crystals of the soaps, is to be considered as abnormal. Gentle heating of the slide will dissolve the crystals and flakes of the fatty acid and soap. Triple phosphate crystals, colorless and of

characteristic shape, neutral phosphate of lime crystals, colorless, or the yellow calcium salts (sometimes bile stained) are commonly found. Oxalate of lime crystals are usual in a mixed diet. Their presence in the fæces when no vegetables are being eaten is said to indicate some intestinal disorder. Cholesterin crystals, Charcot-Leyden crystals, especially if much mucin is present, are both found in the fæces. Very frequently present are the so-called "yellow bodies": large lumps of bright yellow material, structureless, often surrounded by mucus, and recognized macroscopically. They give a proteid reaction. They are considered to be albumin, bile stained, and, when in great amount together with much mucus, indicate some disturbance of proteid digestion.

Casein flocculi are seen microscopically as almost structureless masses, finely fibrillated and enclosing fat droplets in their meshes.

Hairs, cotton and linen fibres, are common in the stools, being taken in with the food in large numbers.

Chemical Examination.—In health the fæces have a neutral or faintly alkaline reaction. On standing this becomes faintly acid. Stools with excess of carbohydrates ferment and give a strong acid reaction. Excess of fat, fatty acid, gives faintly acid stools. Decomposition of excess of proteid matter produces an alkaline reaction. A mixed diet in health causes neutral fæces; a pure proteid diet produces alkaline fæces; a pure carbohydrate diet produces acid fæces; a diet of fats produces acid fæces. Only freshly passed fæces can be used in testing. A markedly acid reaction in fresh fæces suggests fermentative changes from undigested carbohydrates. "Acid diarrhœas," so-called, may be associated with hyperacidity of the stomach and insufficiency of the biliary and pancreatic secretion.

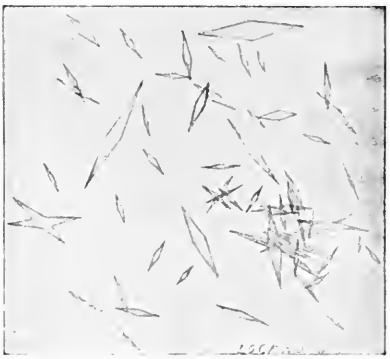


Fig. 91.—Charcot-Leyden crystals from the stools. × 400.—Emerson.

The test for hydrobilirubin or the bile products is important, since they may be present in colorless stools. The fæces are stirred up with a concentrated solution of mercuric chloride; normal fæces are colored red; fæces containing unchanged bilirubin become green. The pale stool of the leucohydrobilirubin gives the red reaction. Absence of the red or green coloring is seen in fatty stools with complete acholia.

Composition.—From 74 to 84 per cent. of the fæces is water; 16–26 per cent. is dry substance. Of the dry substance 10–20 per cent. can be extracted with ether, *i.e.*, are fats. Over 90 per cent. of fats taken in are absorbed.

Fats.—Qualitative tests only can be considered. The fats are readily detected macroscopically and microscopically. They are excreted as neutral fats, soaps, and fatty acids. These have been described. Crystals and flakes melt readily. Extracting a small mass of fæces with ether and pouring the ether through a piece of filter paper will, if fats are in excess, give the characteristic appearance of oil on the paper.

From 2 to 6 per cent. of the dry substance is carbohydrate, usually dextrin. Tincture of iodine or Lugol's solution will stain unaltered starch blue; dextrin remains red. There is no reaction for sugar. Fermentation is the simplest test for excess of carbohydrate or carbohydrate residue. Schmidt's fermentation tube may be employed or one may note carefully the presence of gas formation in the freshly passed stool.

Proteids.—More than 85 per cent. of proteids taken into the body are absorbed. The proteid residue in health is partly from the food, partly from the disintegration of proteids of the body—leucin, tyrosin, indol, skatol, mucin, nuclein. Albumin and globulins, and their transformative products, albumoses, peptones, are not found normally. Their presence in the stools means either insufficient proteid digestion and absorption, or that the "postdigestive putrefaction" in the large intestine has not had time to take place. The simple tests for albumin and albumose can be applied after dissolving, mixing a small amount of fecal material in water, and filtering. Any inflammatory condition of the lower bowel will yield albumin in the fæces. Serous exudation higher up may undergo the natural digestive processes. Persistent intense diarrhœa, choleraic diarrhœa, can hurry materials through before digestion of albumin or albumoses has progressed, and fæces from these conditions may give albumin reactions from food taken or from serous exudation into the bowel as in typhoid fever, cholera, or dysentery.

"Total nitrogen" estimations are necessary to determine the relation of proteid output and intake. As of the fats and carbohydrates one can say of the proteids—for clinical purposes macro- and microscopical examinations yield more useful information.

Digestive ferments are not found. The pigmentary remains of the bile have been spoken of. Mention has been made of the various salts and crystals, phosphates, oxalates, cholesterin, etc., visible microscopically, remains of food digested, or of digestive procedure. Chemical tests show presence of bile salts, bile acids, leucin, tyrosin, xanthin, carnin, and proteid derivatives.

Occult Blood.—The most important chemical examination for practical purposes. Teichman's acid-hæmin test may be used, but others are simpler and more certain. They all depend upon altered hæmoglobin reactions. No examination of fæces can be considered complete unless a blood test has been made; since occult bleeding may go on indefinitely with no gross signs of blood in the fæces and no blood-corpuscles to be seen microscopically. All bleeding from the nose, gums, pharynx, lungs, and vagina must be excluded. No meat can be taken during the days on which the fæces are tested. It is best to wait for forty-eight hours or to mark a food period by giving charcoal, lycopodium, or carmine.

To perform the test we must first remove gross fat by shaking with ether; otherwise the final ether extract may be clouded. This is poured away and the residue is used. 10 c.c. of fluid fæces or 5 c.c. of solid fæces, broken up in 5 c.c. water, are treated with 3 c.c. glacial acetic acid, thoroughly mixed and shaken. This dissolves red blood-cells and sets free hæmoglobin or makes acid hæmatin. After standing a few minutes excess of ether, 20–30 c.c., is added and the mixture vigorously shaken and then allowed to separate. The overlying ether is poured off and the tests made with it as follows:

Turpentine-Guaiac Test.—To a few cubic centimetres of the above ethereal extract previously treated with a little alcohol are added 10 drops of freshly made guaiac tincture and 30 drops of turpentine. In the presence of blood pigment a distinctly blue color occurs. Sources of error are the recent eating of potatoes or other starchy food, iron as a medicine, or the presence of bile, saliva, milk, pus in considerable quantities, and urobilin. The reaction may fail in the presence of minute traces of blood.

Aloin Test.—Klinge and Shaer.—This test is extremely delicate. Foods containing hæmoglobin and all vegetables and drugs must be avoided for several days. The diet period must be determined by charcoal or lycopodium, not carmine. From 1 to 1.5 c.c. of turpentine are superimposed and then 0.5 c.c. of freshly made 3 per cent. aloin solution. The reaction consists in the rapid development at the line of contact of a bright rose-red color. In a doubtful case both these tests may be used.

Benzidin Test.—Schlesinger and Holt's Modification.—1. Concentrated Benzidin Solution: as much benzidin (Merck's benzidin puriss.) as will go on tip of table knife in 2 c.c. of glacial acetic acid; shake lightly. 2. Piece of fæces the size of a pea (or several c.c. of a distilled-water extract) suspended in one-fifth of a test-tube of water; close with cotton and boil. 3. Ten to twelve drops of benzidin solution put in test-tube and 2–2.5 c.c. of hydrogen peroxide (3 per cent.) added. To this add 1–3 drops of boiled fæces after mixing the latter by slightly shaking. Green to blue color is positive and appears in two minutes in a blood mixture of 1–200,000 strength; hence this test is 5–10 times as delicate as other tests.

In ulcerating carcinoma ventriculi, occult blood is continually present in the stools; in ulcus ventriculi there are intervals in which no occult blood can be detected; in intestinal tuberculosis it is absent; in enteric fever it may occur in the absence of gross hemorrhage or may antedate the latter by twenty-four or forty-eight hours.

IV.

THE EXAMINATION OF THE UPPER AIR-PASSAGES AND THE EAR.

RHINOSCOPY. LARYNGOSCOPY. OTOSCOPY.

General Considerations.—Local affections of the nose, throat, or ears may give rise either to local or constitutional symptoms, while constitutional diseases frequently produce local manifestations. For this reason dexterity in the use of the mechanical means by which we are enabled to distinguish between the manifestations of local and constitutional diseases, as observed in these organs, is of no less importance to the general clinician than to the specialist.

The instruments employed in a simple examination of the nose, throat, larynx, or ear are a head mirror for reflection of light, tongue depressor, laryngeal mirror, and nasal and aural specula. They are of varied designs, but any instrument to which the physician has become accustomed will

usually meet the requirements of ordinary cases. Of far greater importance than the instrument to be employed is its careful manipulation. Every instrument must be carefully cleansed in the presence of the patient, both before and after using. The speculum should be slightly warmed over a spirit flame or gas burner before introduction into the nose or ear.

Either natural or artificial light, if sufficiently strong, can be condensed and reflected by the mirror to the point or area to be examined, and the source of the light may be either to the right or left of the patient.

The Examination of the Nose.

Anterior Rhinoscopy.—Excoriations around the margin of the nares are usually produced by acrid secretions, excessively acid or alkaline, which occur in the course of various infectious diseases, colds, nasal hydrorrhœa, syphilis, etc. Rhinoscopy has to do with the examination of the interior of the nose, for which purpose it is necessary to dilate the nostrils, one at a time, with a bivalve speculum, using care to avoid injury to the mucous membrane or unnecessarily annoy the patient by overdistention.

Structures Observed.—Under normal conditions, the patient sitting erect before the operator, with the head tilted slightly backward, the distended alæ should present clearly to view the lower turbinates, the middle and lower meati on the outer walls, the area opposite to these on the septum, and the floor. This constitutes about the lower third or respiratory portion of the nares. The area just within the nares on the lower anterior margin of the septum should be especially examined as the most frequent location of the source of hemorrhage.

Tilting the patient's head backward brings into view the upper or olfactory portion of the nostrils, the middle turbinate and superior meatus, rarely a small portion of the superior turbinate—the close proximity of the septum and outer wall preventing an exposed view of the ethmoid and sphenoid area, superior turbinate and points of entrance to the frontal sinus. It is this space that we frequently find bathed in pus in the case of purulent sinusitis, ethmoiditis, or antrum disease. As a rule, an accumulation of pus above the middle turbinate is an indication of disease of the ethmoid or frontal sinus, while if pus collects beneath the middle turbinate its source is probably from the antrum of Highmore. In case the whole naris is bathed in the purulent secretion, first cleanse the nostril, then have the patient lean forward or turn the head well toward the side involved in order to favor the discharge of fresh pus and determine its origin more clearly. Nasal polypi most frequently originate in this part of the nares, at the marginal mucosa of a turbinate which has undergone mucoid degeneration from necrotic tissue in the ethmoid cells; less often from the sphenoid sinus, which lies slightly below and posterior to the ethmoid cells. Beneath the middle turbinate is the only natural opening into the antrum of Highmore—the *ostium maxillare*—which, however, in many cases is so obscure as to be found with difficulty even by experienced rhinologists. Occasionally two or more openings enter the antrum at variable points, even as high as the floor of the orbit. The inferior meatus is important for two reasons: first, it is beneath the lower turbinate that we find the

nasal opening of the lachrymal duct, which may become occluded from either an acute or chronic enlargement of the turbinate; second, because of the thinness of the bony wall dividing the nares from the antrum of Highmore, through which a cannula may be easily introduced for diagnostic purposes in suspected purulent infection of the sinus.

If on first looking into the nose the view is obstructed by an intumescent condition of the membrane, which is found in nearly every local congestion, whether active or passive, the difficulty of obtaining a satisfactory view will be greatly obviated by the introduction of a small pledget of cotton dipped into a solution of cocaine and camphor, each two grains to the ounce of liquid albolene. The objection to the adrenalin preparations in examination is threefold: first, it frequently acts as an irritant, throwing the patient into a violent state of sneezing; second, by the intense

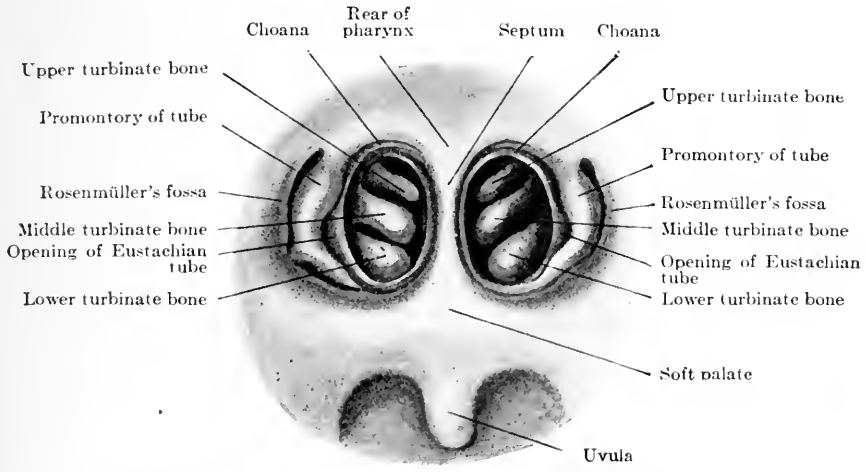


FIG. 92.—Normal posterior nares, view obtained by repeated change of the mirror.

bleaching of the membrane; and, thirdly, because of the aggravated congestion which follows its use. All the accessory cavities herein referred to are in direct communication with the nares; each sinus or cell is lined by mucous membrane, somewhat modified in character from that in the nasal chambers, and any inflammatory process in one cavity may cause more or less irritation in one or all of the others.

Posterior Rhinoscopy.—To examine the nasopharynx there are needed a head mirror, tongue depressor, and rhinoscopic mirror. Some persons are able to depress their tongues by voluntary muscular effort, in which case the depressor is not needed. There is also a great difference in the ability of individuals to relax the soft palate at will, thus allowing an unobstructed vision in the mirror of the vault of the pharynx and the posterior nares. The process of such examinations will often require great patience if the pharynx be hypersensitive, since the slightest touch with the mirror may produce gagging.

Let the patient sit comfortably in the chair and assure him that there will be nothing connected with the examination to cause either pain or

discomfort. There is a general tendency to hold the breath and strain on the pharyngeal muscles. To obviate these difficulties explain that it is important to allow the mouth to open widely and easily, without the slightest tension of the jaw, leaving the tongue at rest in its natural position, and to breathe quietly and freely through the mouth. The probability is that after this reassurance the soft palate will relax to its normal position. A common difficulty consists in the involuntary retraction of the soft palate tightly against the pharyngeal wall as soon as the mirror approaches the mouth, and its retention in that position until the mirror is withdrawn. This frequently can be obviated by having the patient close his eyes. Should this fail, the most satisfactory recourse left is cocainization to a degree sufficient to relieve the hypersensitiveness, when with a long applicator, bent at right angles, making a hook about three-fourths of an inch long on the end, the soft palate may be gently drawn forward, and the rhinoscope placed in position to reflect the image desired. It is always better to twist a small piece of cotton on the retractor, which being dipped into a bland oil will prevent injury to the mucous membrane.

Structures Observed.—With the rhinoscope just below and posterior to the margin of the soft palate, and with a strong light, the angle of reflection in the mirror may be so directed by manipulation as to show successively all the structures in the nasopharynx, viz., the Eustachian orifices on the extreme outer margins, and just above and slightly posterior to these the fossæ of Rosenmüller, which are occasionally obstructed by adhesive bands; in each naris are seen the middle and lower turbinates, the latter being indistinct except over its upper half; and directly posterior and below the posterior margin of the septum on the pharygneal wall is the usual position of the pharyngeal tonsil or adenoids. Since this lymphoid structure under normal conditions undergoes atrophy about the age of puberty, when observed in adults, or when sufficiently large in children to interfere with nasal respiration, it should be regarded as pathologic. Polypoid growths in the nasopharynx originate usually from mucoid degeneration of the posterior margins of the middle or superior turbinates or from the posterior ethmoid cells; fibromata, sufficiently large to fill the entire vault, suspended by a small pedicle and hanging low enough in the oropharynx for the lower margin to be seen by direct vision, are not infrequently observed. Posterior rhinoscopy is seldom accomplished in children with any degree of satisfaction, in which case ocular inspection must be supplanted by digital examination.

Laryngoscopy.

For the examination of the laryngopharynx, larynx, and trachea the same instruments are required as those used for posterior rhinoscopy, and the same precautions toward preventing nervousness on the part of the patient during examination are of even greater importance. The tongue depressor will not be needed in all cases, since in some a better view can be obtained by grasping the tip of the tongue with a towel or handkerchief and drawing it well out and downward, using care not to

cause pain underneath the tongue by too forceful traction over the lower teeth. In still others the patient may be able voluntarily to depress the tongue.

The oropharynx is examined by direct inspection. The appearance and color of the mucous membrane of the posterior pharyngeal wall vary greatly according to the condition of the gastro-intestinal tract. The redness frequently observed along the anterior borders of the faucial tonsillar pillars in gouty or lithæmic individuals is a sign of diagnostic importance. This may vary in color from a dark pink blush to a purplish crimson, and may be regular in outline or occasionally present the appearance of petechial spots, particularly on the uvula. Another phenomenon often observed is indicative of either acute or chronic inflammatory Eustachian or middle-ear involvement. It consists of a prominence or bulging of the postpharyngeal wall, evidently an inflammatory infiltrate, just back of the posterior faucial pillar on the same side as that of the affected ear.

In the examination of the laryngopharynx the laryngoscope is used. Observe the base of the tongue carefully to detect the presence of an enlarged lingual tonsil, which gives rise to various annoying symptoms, most prominent of which is the constant accumulation of mucus about the glottis and the resulting pharyngeal tenesmus. Occasionally this mass of tonsillar tissue is sufficient to press the epiglottis downward and thus interfere with the examination of the larynx proper.

Foreign Bodies. — The most frequent locations of foreign bodies, such as broken bits of toothpicks or match-sticks, fish-bones, tooth-brush bristles, etc., in the laryngopharynx are the glosso-epiglottidean pouches at the base of the tongue, or else in the sinus pyriformis which lies partially posterior to and on either side of the glottis. The patient's sensation of locality of a foreign body in such a position is frequently misleading; for instance, a fish-bone or bristle sticking in the base of the tongue may give the sensation of being farther down in the larynx, or perhaps even in the nasopharynx.

Examination.—A strong, well focussed light is essential, and whether the patient be in the sitting or recumbent position, the head must be well extended and free breathing through the mouth insisted upon. The autoscope, an instrument devised some years ago for the purpose of making direct inspection of the larynx, is not generally employed at the present time. Proceeding with the usual method, the patient's tongue is depressed, or drawn outward, the laryngoscope is carefully introduced into the upper laryngopharynx in a manner that will push the uvula backward out of range of the reflected laryngeal image. The best angle of reflection can be determined according to the case in hand, the epiglottis, owing to its variability both in point of shape and position in different individuals, being the principal obstacle to a clear view of the underlying structures. This difficulty, however, can best be obviated by the influence which the effort on the part of the patient to produce certain vocal tones has upon the position of the larynx. Two vocal sounds are utilized; first, the classic "ah," during the intonation of which the larynx is in the most natural relation to the surrounding structures at rest, except for the fact that the cords are approximated or in the position of phonation. With the parts

in this position there will be reflected in the laryngoscope the edge of the epiglottis and a narrow margin of its underlying surface, the arytenoids, and the posterior half of each vocal cord, which appears in the mirror as the inferior half.

The same relation will still be preserved if the patient now be instructed simply to breathe, allowing the arytenoids and hence the cords to swing freely open. But to obtain an image of the junction of the cords at the anterior ends, appearing superiorly in the mirror, the effort to produce the vowel tone "e" must be made. This will so elevate the larynx and change its position in relation to the epiglottis and other structures as to expose the whole length of the cords and the whole inferior surface of the epiglottis in one view, and in most cases, after holding the tone for a few seconds, the patient may breathe freely without the tongue falling back to its original position. A good plan is to have the patient hold the note for a moment, followed by free respiration, and repeat the process as often as required till a satisfactory view is obtained of all the intralaryngeal structures. During respiration the anterior wall of the trachea also may be seen, in some cases as far down as the bifurcation, though to be satisfactory an examination of the lower part of the trachea and bronchial tubes should be made with a bronchoscope. This instrument has been perfected in recent years to such an extent as to be of great value in the hands of a skilful operator for the removal of foreign bodies or for the inspection of any diseased condition of the lining membrane. If during the examination the patient has an inclination to gag, free and rapid respiration may overcome it; should the tendency persist, however, withdraw the mirror and allow the throat to be at rest for a short time; under no condition will anything be gained by forcing or attempting to prolong an examination when the patient coughs, gags, or the muscles of the throat become fatigued.

The larynx is subject to the same inflammatory changes which may take place in any other mucous membrane, and likewise to any local infection. The histologic structure of the submucous tissue seems to favor rapid and extensive œdema from local inflammations, due to traumata, scalds, and the inhalation of irritant vapors; from infectious processes involving adjacent structures, as diphtheria, follicular tonsillitis, and tuberculosis; and from circulatory disturbances such as may arise from cardiac or renal lesions.

Chronic hoarseness not amenable to treatment, particularly in individuals past forty years of age, must be regarded as suspiciously indicative of malignancy and be kept constantly under observation in order that should such a condition exist it may be detected at the earliest stage possible. Sluggishness in the movement of the vocal cord, or even an apparent paralysis of the cord on the affected side, has been observed not infrequently in laryngeal carcinoma long before any actual tumor was visible.

Otoscopy.

For convenience in description the organ of hearing is usually divided into the external, middle, and internal ear. The last embraces that part of the petrous portion of the temporal bone in which the terminal fila-

DESCRIPTION OF PLATE III.

1. Laryngeal image during respiration.
2. Laryngeal image during phonation.
3. Laryngoscopic picture in a case of paralysis of the right recurrent laryngeal nerve.
4. Laryngoscopic picture in a case of bilateral paralysis of the recurrent laryngeal nerves.
5. Laryngoscopic picture in a case of paralysis of the interarytenoid muscle.
6. Position of the vocal cords in unilateral adductor paralysis.
7. Position of the vocal cords in bilateral adductor paralysis—during efforts at deep inspiration.
8. Position of the vocal cords in paralysis of the right internal tensor.

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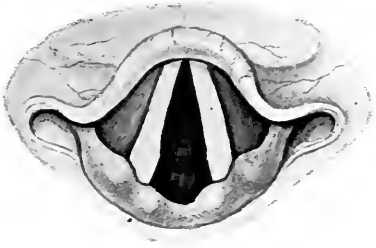

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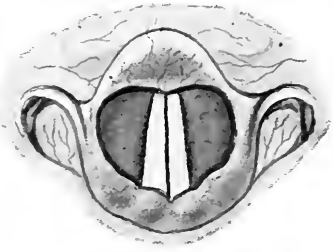

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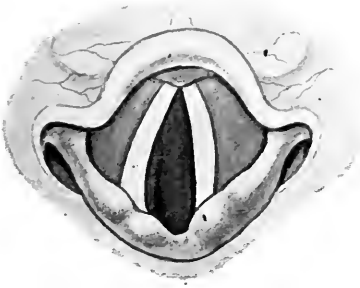

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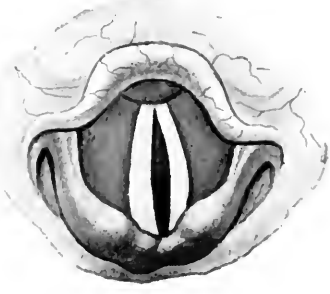


ments of the auditory nerve are distributed, and therefore is also designated as the sound-perceiving apparatus. The external and middle ear, since they serve the purpose of transmitting sound impressions to the nerve, are called the sound-conducting apparatus.

It is of importance to distinguish between diseased conditions of the sound-perceiving and the sound-conducting apparatus, or between disturbance of hearing caused by nerve lesions and that dependent upon diseased structures of the ear itself. For example, in any case of deafness the first thing to be ascertained is what part of the ear, if any, is at fault. Deafness, either partial or complete, may be caused by obstructions in the external auditory canal, such as foreign bodies, impacted cerumen, congenital atresia, exostosis, furunculosis, etc., and also by hemorrhage into the semicircular canals, or as the effect of certain drugs. The condition of the external canal and the tympanic membrane can easily be determined by direct ocular inspection, a strong, well focussed light being directed into the canal through a suitable speculum. If the canal be found clear, then the difficulty must lie either in the middle or the internal ear. To distinguish between these the tuning-fork test, devised by Weber, is usually employed.

External Auditory Canal.—The external auditory canal varies greatly in size and somewhat in direction in different individuals. The cartilaginous portion of the canal is usually directed more or less downward and forward, so that in order to bring this part of the canal and the bony meatus into the same axis for inspection of the walls of the canal and the drum membrane it is necessary to draw the auricle gently upward and backward. By holding the auricle in this position with one hand and manipulating the speculum with the other—a metallic conical speculum is the most desirable—every part of the canal wall and drum membrane may be clearly seen. Note the size of the canal and any acute inflammatory swelling or chronic induration. The cartilaginous portion of the canal comprises a little over one-third of the whole length of the meatus. Its junction with the bony meatus is the most frequent site of furunculosis. In young children the cartilaginous meatus comprises about two-thirds of the whole extent of the canal. When a furuncle is of deep origin pus may burrow beneath the periosteum inward toward the tympanic cavity, occluding the osseous meatus entirely and giving rise to most excruciating pain. The pain within the ear and swelling extending even back of the auricle may be confused with acute mastoiditis. In furunculosis the most acute pain is apt to be elicited by pressing upon the tragus, or, if there be postauricular tenderness, it will likely be superficial; in mastoiditis, however, the pain may be slight superficially and intensified by deep pressure over the mastoid, and pain is not apt to be elicited on pressure over the tragus.

When the cartilaginous portion of the canal is occluded by swelling, gently insert a tightly rolled pledget of cotton dipped in a solution composed of camphor and carbolic acid, equal parts, and allow it to remain a few minutes. The swelling is thus sufficiently reduced to allow the introduction of a small speculum for the examination of the deeper canal and tympanic membrane. This solution also produces partial anæsthesia of the membrane, thus allowing a more thorough examination.

The tympanic membrane separating the external canal from the tympanum, irregularly oval in shape and slightly concave in its normal state, is affected to some degree by every inflammatory disease of the middle ear, both acute and chronic, and should therefore receive most careful attention in every aural examination. A strong, well focussed light is a necessity and the largest speculum which the canal will admit should be used. The external layer of the drum membrane is modified skin, clear and almost translucent in its normal condition, and through it can be seen the impression of the malleus with which it lies in direct contact. Acute inflammations of the middle ear produce a pink or reddish hue along the margins of the malleus and in some cases over the entire membrane.

Exudates, serous or purulent, in the tympanic cavity, even though small in quantity, produce bulging of the membrane and frequently terminate in spontaneous rupture into the external canal. In acute and chronic inflammations causing occlusion of the Eustachian tube the tympanic membrane will be found retracted. Retraction may also be brought about by adhesions within the tympanic cavity following marked inflammatory involvement. In a case of retracted membrane we can ascertain whether the tube is patulous by one of the usual methods of inflation. Valsalva's consists of a vigorous expiratory effort while the nose and mouth are kept closed. Politzer inflates the tympanum through one nostril by compression of a rubber air-bag while the patient is in the act of swallowing. The opposite nostril and the mouth are closed. Eustachian catheterization is the most satisfactory method in difficult cases. With Siegel's otoscope the air within the external auditory canal can be exhausted and adhesions involving the tympanic membrane observed. Aural polypi originate most frequently within the middle ear from granular or necrotic tissue and protrude into the external canal through perforations in the tympanic membrane, though they occasionally may be found in any part of the canal, particularly at the cartilaginous and osseous junction. Exostoses occur in any portion of the osseous canal, particularly from the posterior wall and from the osseous and cartilaginous junction. In chronic non-suppurative processes involving the **middle ear,** the drum membrane becomes opaque and thickened, and usually distorted in shape. In cases of otosclerosis white chalky spots are observed in the membrane which may otherwise appear normal. A sign of diagnostic importance in mastoiditis complicating chronic suppurations of the middle ear is an infiltration of the membrane covering the superior posterior osseous wall of the external auditory canal, presenting the appearance of a circumscribed drooping or bulging.

Pharynx and Eustachian Tube. — No examination of the ear can be considered complete without a careful inspection of the nasopharynx at the entrance of the Eustachian tube slightly below and anterior to the fossa of Rosenmüller. The technic of this procedure is described under posterior rhinoscopy. Catheterization of the tube for diagnostic purposes can be accomplished either through the nose or by way of the oropharynx.

V.

THE EXAMINATION OF THE BLOOD.[1]

General Considerations. — Information derived from blood examinations, while not essential in the establishment of a diagnosis in most instances, is frequently a useful aid. Negative blood reports are often important in diagnosis, as in the exclusion of malaria from a group of diseases which have similar clinical features, such as malignant endocarditis, septicæmia, and certain types of tuberculosis. Diseases associated with marked splenic or glandular enlargement present so close a resemblance to leukæmia that only a study of the blood can exclude the latter condition. Many blood examinations elicit results which assist in arriving at or completing a diagnosis. Evidence of a pathognomonic character gained from hæmatological studies is available in only a limited number of diseases, notably in myelogenous leukæmia, malaria, relapsing fever, trypanosomiasis, and filariasis. The condition of the blood as to hæmoglobin value, the number of erythrocytes and leucocytes, may serve as an index of body nutrition. Blood counts often yield information which bears upon prognosis,—*e.g.*, in chlorosis a steady hæmoglobin rise is an evidence of favorable progress of the patient, while an erythrocytic gain in progressive pernicious anæmia or leucocytic decrease in leukæmia likewise points to improvement. Counts of the white corpuscles also aid in establishing the leucocytic standard of the patient. The opsonic index and the agglutination phenomenon are recognized adjuncts in the field of diagnosis.

Methods of Blood Examination.

Obtaining Blood. — For most clinical examinations a few drops of blood, obtained from a puncture in the lobe of the ear or the finger-tip, will suffice. The lobe of the ear is sometimes selected for making the puncture on account of its lessened sensibility and because the operation can be performed without the patient seeing it, but the finger-tip is generally chosen as this site is more convenient for the examiner. The puncture should be made with a lancet-shaped or triangular surgical needle, an instrument especially devised for this purpose, or a steel pen with one of the nibs broken off. The part selected should be cleansed with alcohol or with soap and water followed by alcohol, and dried with a towel or handkerchief. If not warm, the skin is warmed by gentle friction, but forcible rubbing should be avoided, since it excites active hyperæmia. If the individual is a bleeder, the precaution of making a superficial puncture and of having measures at hand to control hemorrhage should be observed. It is obvious that areas of œdema and of inflammation must be avoided. If the former be present about the hands or ear, an area free or nearly so of œdema is chosen. The puncture is made with a quick thrust of the instrument, which has previously been cleansed with alcohol or passed through a flame. The first drop or two of blood should be wiped away.

[1] Originally contributed by Prof. Kalteyer. Revised by Dr. Turner.

Forcible squeezing of the tissues in the immediate vicinity of the wound must be avoided, as this may alter the composition of the blood by the addition of lymph fluids. As the blood flows from the wound, its gross appearance as to color and fluidity is noted.

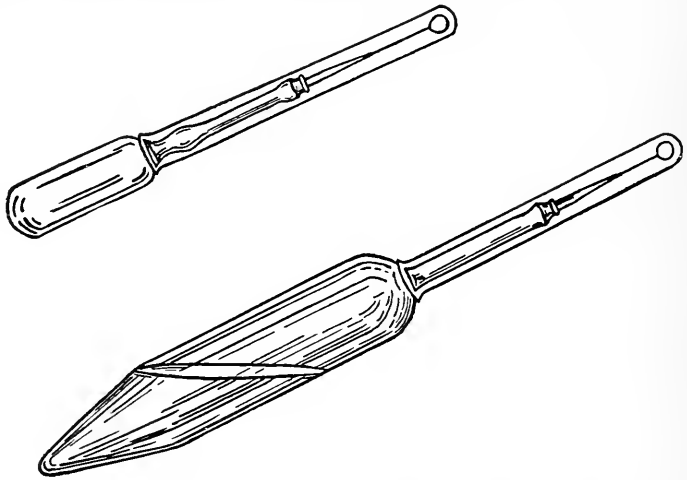


FIG. 92*a*.—Vacumm syringes for collecting blood for examinations.

The blood may be drawn from a vein at the elbow when larger quantities are required for extensive physical, chemical, and biological examinations. A tourniquet or bandage is applied to the upper arm just firmly enough to obstruct the venous return but not so tight as to eliminate the arterial pulse. The tourniquet is often removed before the blood is allowed to flow in order to eliminate any alteration caused by congestion. The sterile needle of a syringe is plunged into the vein after the part has been surgically cleaned. The required quantity is thus readily obtained.

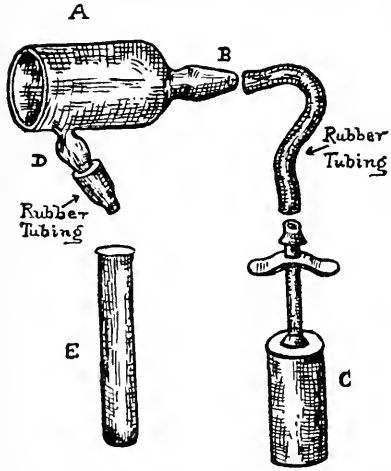


FIG. 92*b*.—Suction apparatus for collecting blood for the Wassermann Reaction. *A*, suction glass connecting at *B* with suction pump *C*; the blood flows through *D* connected by rubber stopper with an ordinary test tube, *E*.—Blackfan.

Blackfan has perfected a very simple and inexpensive apparatus for use in collecting blood—from infants and older patients—for serological examinations, although not of value in obtaining blood for other examinations since there is an admixture of tissue fluids with the blood. The apparatus is connected for use. The part selected is usually below the angle of the scapula. A small puncture is made in the sterilized skin in one or more points and the apparatus immediately applied. The necessary amount is easily secured without the use of much suction. When the blood has been withdrawn the area is covered with a sterile dressing.

Preparation of Fresh Blood for Immediate Examination.—A cover-glass is applied to a droplet of blood and then placed upon a clean slide. The blood usually spreads into a thin layer. Warming the slide by friction with a piece of gauze, a handkerchief, or tissue paper before applying the cover-glass facilitates spreading. In a well-prepared preparation the corpuscles are arranged in a single layer separated from each other over an area sufficiently large for the desired study. If it be necessary to delay the examination, drying of the specimen can be prevented by ringing the margins of the cover-glass with vaseline or cedar oil.

Preparation of Blood for Staining.—Cover-glasses which have clean polished surfaces are placed upon a sheet of paper, or preferably upon a folded towel, from which they can be picked up easily. A cover-glass, held with the fingers, or with forceps, is applied to the summit of the droplet of blood, being careful to avoid touching the skin, and allowed to fall upon another cover. As soon as the blood has ceased spreading, the covers are slid apart. In performing this operation, care should be exercised not to lift the glasses apart: the sliding motion must be performed rapidly, avoiding a jerky, uneven stroke. The smear may be made upon a slide by placing a drop of blood upon it and spreading with another slide or with a glass rod especially designed for this purpose.

Methods of Fixation.—Heat Fixation.—The covers are placed in an oven, the ordinary dry-heat sterilizer being convenient for this purpose, and heated gradually until the temperature reaches to 120° C. or up to 155° C. and subjected to this temperature for from ten to twenty minutes. A convenient plan for fixing the specimens with heat consists in placing the spreads upon a heated copper plate. The plate, about 20 centimetres in length, 8 centimetres in width, and from ½ to 1 centimetre in thickness, supported by a suitable stand, is heated at one end with the flame from a Bunsen burner or an alcohol lamp. When the plate is thoroughly heated, the covers are placed upon it at a point where the temperature is sufficient to boil water (which is previously determined by dropping water upon its surface, beginning at the end farthest away from the flame) and exposed to this heat for about thirty minutes. A method less suitable than the ones mentioned consists in passing the film rapidly through a Bunsen flame forty or fifty times.

Fixation by Wet Methods.—Fixation may be obtained by submerging films in a mixture of equal parts of absolute alcohol and ether for twenty or thirty minutes, or in absolute alcohol for five minutes. The Futcher-Lazaer method subjects the films to .25 per cent. formalin in 95 per cent. alcohol for one minute: they are then rinsed in water and dried with filter-paper.

Blood Staining.—Many methods for staining blood are available. To Ehrlich belongs the credit of devising a mixture by which all known varieties of blood-cells except those which contain basophilic granules are completely colored. At the present time certain panoptic fluids containing eosin-methylene-blue compounds are employed extensively in routine work, having largely supplanted Ehrlich's triple stain.

Ehrlich's triple stain is prepared by mixing saturated aqueous solu-

tions of acid fuchsin of orange G. and of methyl green 00 with glycerin, ethyl alcohol, and water.

Staining Technic.—The stain is applied to the blood-film previously fixed by heat, for five minutes, after which the excess of stain is drained off and the cover washed with water, dried, and mounted in xylol balsam or cedar oil. Normal erythrocytes are colored orange, eosinophilic granules dull red, neutrophilic granules violet or lilac, nuclear structures various shades of green, blue, or black, malarial parasites and bacteria green or blue, while basophilic granules are unstained.

JENNER'S STAIN is prepared as follows: Mix equal parts of a 1 per cent. aqueous methylene-blue solution with a 1.25 per cent. aqueous eosin (water soluble) solution. After shaking thoroughly, the solution is allowed to stand for twenty-four hours and then filtered. The precipitate is dried. One part of precipitate is dissolved in two hundred parts of methyl alcohol. Films are treated with this solution without previous fixation for from three to five minutes, washed with water, dried, and mounted in xylol balsam or cedar oil. The following tinctorial reaction is secured: Normal erythrocytes stain terra-cotta; nuclei, various shades of blue or green; basophilic granules, dark blue; neutrophilic granules, pink; eosinophilic granules, bright red; the cytoplasm of lymphocytes and malarial parasites, a deep blue. A deposit of dark granules upon the film which is often observed is an objectionable feature and interferes with the usefulness of this method.

LEISHMAN'S STAIN, an improvement on Jenner's, is based on the Romanowsky method. It is prepared as follows: (1) A one per cent. aqueous solution of methylene blue (Gruber's medicinal), containing 5 per cent. of sodium carbonate, is heated at 65° C. for twelve hours and then allowed to stand for ten days. (2) An equal volume of a 1 per cent. solution of eosin in distilled water is added to the methylene-blue solution in an open vessel and the mixture stirred from time to time. After twelve hours the resultant sediment is collected on filter-paper and washed with water until the washings are almost colorless. One and a half parts of dried powdered precipitate are added to one hundred parts of pure methyl alcohol. Three or four drops of this stain are placed upon the unfixed blood-film and allowed to act for thirty seconds, when double the amount of water (six or eight drops) is poured upon the cover and mixed with the stain. After five minutes the spread is washed gently with water and a few drops allowed to remain upon the specimen for about a minute. The smear is now dried, first between filter-paper and then in the air, and mounted in balsam or cedar oil. The nuclei of leucocytes and of erythroblasts, and blood-platelets are stained various shades of purple, the protoplasm of lymphocytes and certain polychromatophilic erythrocytes various tints of blue, basophilic granules dark violet or royal purple, normal erythrocytes and eosinophile granules pink, and neutrophile granules a dull red. Malarial parasites and trypanosomes are distinctly stained by this method.

WRIGHT'S STAIN contains an eosin-methylene-blue combination held in solution by methyl alcohol. The unfixed film is stained for one minute, next 8–10 drops of distilled water are added to film stain and allowed to stand two to three minutes. The film is stained deep blue. It is now

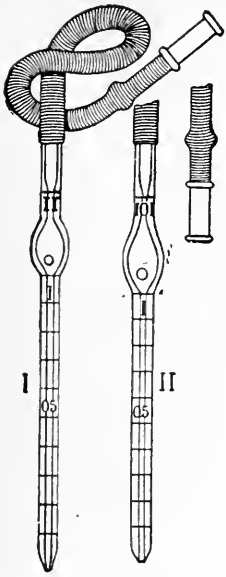


FIG. 93.—I. leucocytometer: II, erythrocytometer of Thoma-Zeiss hæmocytometer.

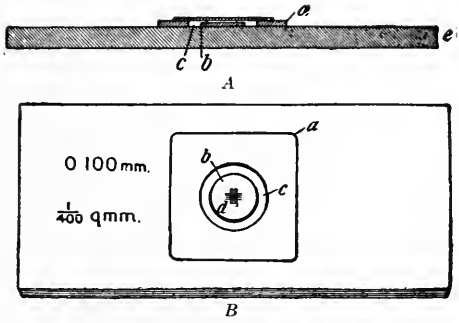


FIG. 94*a*.—Counting chamber of the Thoma-Zeiss hæmocytometer. *A*, profile view; *B*, face view; *a*, wall of cell; *b*, central disk; *c*, groove about disk; *d*, ruled surface

FIG. 94*b*.—Levy counting chamber with double Neubauer ruling.

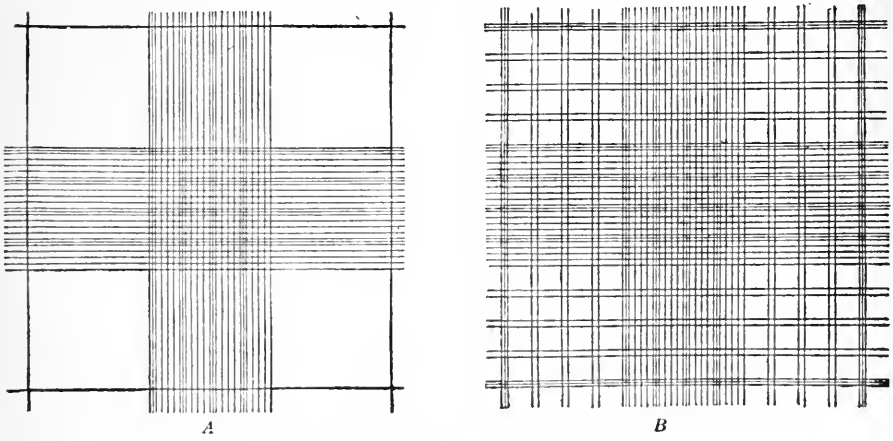


FIG. 95*a*.—*A*, Zappert ruling; *B*, Türk's ruling.

decolorized by washing with water until yellowish or pink. Dry between filter-paper and mount in balsam.

End Result.—Erythrocytes orange or pink; nuclei of leucocytes, blue; neutrophile granules, lilac; coarse mast cell granules, deep purple. The nuclei of erythroblasts and bacteria stain various shades of blue; blood plaques purplish flecked with red. The body of the malarial parasite stains blue. The chromatin varies from blue to red to almost black.

Double Staining.—The films, after suitable fixation obtained by immersion in absolute alcohol, alcohol and ether, or by heating as previously described, are treated first with an acid stain followed by a basic dye, rinsed in water, dried, and mounted. A staining fluid containing acid and basic coloring principles may be employed for this purpose. Most of the methods of double staining are not suitable for differentiating all forms of blood-cells, as certain histological elements remain unstained. Neutrophilic granules are as a rule not colored, and therefore neutrophilic myelocytes cannot be distinguished from large mononuclear leucocytes.

Plehn's stain has the following formula:

Saturated aqueous solution of methylene blue	60 c.c.
One-half per cent. eosin solution (in 75 per cent. alcohol)	20 c.c.
Distilled water	40 c.c.
Twenty per cent. solution of caustic potash	5–1 c.c.

Specimens are fixed in absolute alcohol for from three to five minutes, stained with Plehn's solution, washed in water, dried, and mounted. This mixture stains malarial parasites blue and eosinophilic granules red.

Eosin and Methylene Blue.—A convenient plan consists in treating the fixed smear with a solution consisting of eosin .5 part in 70 per cent. alcohol 100 parts, for a minute or two; wash the cover in water, and then counterstain with a half-saturated solution of methylene blue or Delafield's hæmatoxylin solution for a half to one minute. The specimen is then rinsed in water, dried between bibulous paper or in the air, and mounted.

Chenzinsky recommends a stain composed of 40 cubic centimetres of saturated methylene blue, 20 cubic centimetres of a .5 per cent. eosin solution in 70 per cent. alcohol and 40 cubic centimetres of distilled water. Films fixed in absolute alcohol are subjected to Chenzinsky's solution for from three to six hours, the staining being done at 37° C. in an incubator. *Ehrlich* suggested a mixture consisting of hæmatoxylin 2 grammes, eosin 0.5 gramme, absolute alcohol 100 grammes, distilled water 100 grammes, glycerin 100 grammes, acetic acid 10 grammes, and an excess of alum. The stain is not ready for use until several weeks have elapsed, since this time is required for the ripening of the stain.

Basophilic granules may be demonstrated by a stain recommended by Ehrlich which has the following formula:

Saturated alcoholic solution of dahlia	50 c.c.
Acetic acid	10–20 c.c.
Distilled water	100 c.c.

Differential Counting.—This method consists of determining the relative number of the different forms of blood-cells, generally expressed in percentage figures and sometimes as the number per cubic millimetre. The leucocyte differential estimation is important in the diagnosis of a number

of conditions. An approximate differential count can be made by an examination of fresh, unstained blood by the experienced worker, but for accurate determinations stained films are essential. A mechanical stage is necessary for this method of counting.

Technic.—The specimen is brought into focus, and the slide is shifted with the mechanical stage so as to bring successive fields into view, being careful not to pass over any portion more than once. The different forms of leucocytes are noted and their number recorded until at least five hundred cells have been studied. From these figures the relative percentages are calculated. When nucleated erythrocytes are encountered, their number should also be noted, and the total number of these cells per cubic millimetre can be determined by the following formula:

$$\frac{\text{Number of leucocytes per cu. mm.} \times \text{number of nucleated red cells counted in the stained film}}{\text{Number of leucocytes counted in the stained film}} = \text{Number of nucleated erythrocytes per cubic millimetre.}$$

It is sometimes important to estimate separately the different varieties of abnormal red cells, especially the varieties of nucleated cells.

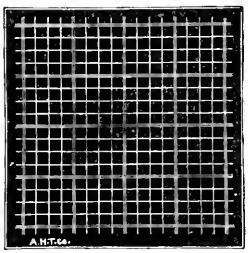


Fig. 95*b*.—Neubauer Ruling for blood counting.

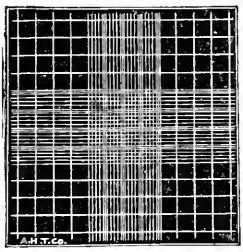


Fig. 95*c*.—Fuchs-Rosenthal ruling for spinal fluids.

Enumeration of the Erythrocytes, Leucocytes, and Blood-platelets.—For clinical purposes, the red cells are counted in a small amount of blood of known quantity, from which an estimate of the number per cubic millimetre is made, this figure being the standard upon which the variations in health and disease are based. A number of methods are available for this purpose. The one recommended by Thoma is generally selected, as it gives fairly accurate results.

The Thoma-Zeiss Hæmocytometer.—This apparatus consists of two graduated pipettes (the red and white counters) for measuring, diluting, and mixing the blood, and a glass chamber in which the corpuscles are counted. The erythrocytometer consists of a graduated capillary tube, upon which the figures .5 and 1 appear. The tube expands into a bulb, above which the figure 101 is inscribed. A rubber tube with a mouthpiece attached is fastened to the short end of the pipette. Filling the pipette with blood to the point marked .5 and then drawing a diluting solution into it until the fluid reaches to the point marked 101, insures a blood dilution of 1 : 200, while a dilution of 1 : 100 is obtained when the pipette is filled to the point marked 1, and then with a diluent to the mark 101. The white pipette, or leucocytometer, is similar in construction to the red pipette, but differs in that the capillary bore is larger and the bulb smaller so that dilutions of 1 : 20 and 1 : 10 may be secured.

The counting chamber consists of a heavy glass slide upon which is cemented a glass plate having a circular opening; a disk is cemented to the slide so that it occupies a central position in the circular opening of the plate. The disk is slightly thinner (by one-tenth of a mm.) than the plate which surrounds it. When the cover-glass, a part of this instrument, is placed upon the plate, the distance between the disk and the cover is one-tenth mm. The surface of the disk is ruled by vertical and horizontal lines one-twentieth of a mm. apart. These lines form four hundred squares, the dimensions of each being one-twentieth by one-twentieth mm. Groups of 16 squares are indicated by a double ruling. The space overlying each square between the surface of the disk and the cover-glass measures $\frac{1}{10\,00}$ cu. mm. ($\frac{1}{20}$ mm. $\times \frac{1}{20}$ mm. $\times \frac{1}{10} = \frac{1}{40\,00}$ cubic millimetre). Zappert's modified ruling of the Thoma-Zeiss counting chamber divides the surface into eight large squares, immediately surrounding the 400 small squares; each large square is equal to the surface ruling of the 400 central squares. The total ruling represents an area of 3600 small squares.

The Levy Counting Chamber is said to possess the following distinct characteristics:

Increased visibility of the rulings when chamber is filled with solution.

It entirely avoids the cemented cell and the attendant danger of its loosening by the drying out of the balsam cement, and the loosening of the ruled counting surface is also greatly reduced.

The parallel form of cell used provides a more uniform distribution of corpuscles over the ruled area and entirely removes the effect of atmospheric pressure upon the depth of the solution, a source of considerable error in the circular form chambers.

The parallel form of cell facilitates cleaning as compared with the circular type.

The matte finish on the surface of the slide insures better approximation between the under surface of the cover-glass and the supporting surface of the slide than when two polished surfaces are used.

Technic of Counting the Erythrocytes.—Special fluids are employed for diluting the blood. *Toisson's solution* stains nuclei a pale blue, therefore rendering differentiation between non-nucleated erythrocytes and white corpuscles easy. Its composition is as follows:

Methyl violet, 5B	0.025	part
Sodium chloride	1.0	part
Sodium sulphate	8.0	parts
Neutral glycerin	30.0	parts
Distilled water	160.0	parts

Hayem's solution:

Mercuric chloride	0.25	part
Sodium chloride	0.5	part
Sodium sulphate	2.5	parts
Distilled water	100.0	parts

Other diluting fluids recommended for clinical work are a 2.5 per cent. aqueous solution of potassium bichromate, a .5 per cent. aqueous solution of sodium sulphate, and a .7 per cent. aqueous solution of sodium chloride. These solutions should be filtered before using.

The blood obtained in the usual manner is drawn into the erythrocytometer to the point .5, unless decided oligocythæmia is suspected, when it is desirable to fill to the mark .1, after which the tip of the pipette is wiped. Toisson's or some other diluting solution is drawn into the pipette until the fluid reaches to the point 101. The pipette should be rotated gently as the diluting fluid enters the bulb, in order to secure a mixture. After filling the pipette, the thumb and finger are immediately placed over its ends and the instrument shaken for about a half minute, in order to obtain a thorough mixture. The unmixed fluid in the capillary portion is then blown out. The counting chamber is now placed upon a perfectly level surface and a droplet of the mixture is deposited in the central portion of the ruled disk. The pipette should be shaken just before adjusting the diluted blood, and the fluid in the capillary portion should always be expelled after mixing in this manner, since corpuscles in the capillary tube may gravitate on standing, thus creating an uneven mixture. The cover-glass is then quickly adjusted in its position. If the fluid flows into the depression surrounding the disk, the operation must be repeated. After the corpuscles have settled, the counting chamber is placed upon the stage of the microscope and a field of 16 squares is brought into focus. In general routine work, the calculation of determining the number of erythrocytes per cubic millimetre is usually based on the number of cells found within 64 squares, provided a uniform distribution of the cells exists. The following plan may be adopted in counting the corpuscles: The cells within the upper left-hand corner square of a group of 16 squares are first counted, then the cells in each of the remaining three squares in that line, going from left to right, after which the corpuscles in the next row of squares are enumerated, proceeding from right to left, next those in the third row and finally in the last line of squares, as shown in the diagram (Fig. 96). The counting chamber is now moved, so as to bring into focus another area of 16 squares, and the number of cells in this group is estimated. This process is repeated until the desired number of squares (not less than 64) has been covered. In order to avoid confusion in counting, the corpuscles which touch the right and lower lines are included in the count of the square in question. The formula for calculating the number per cubic millimetre is as follows:

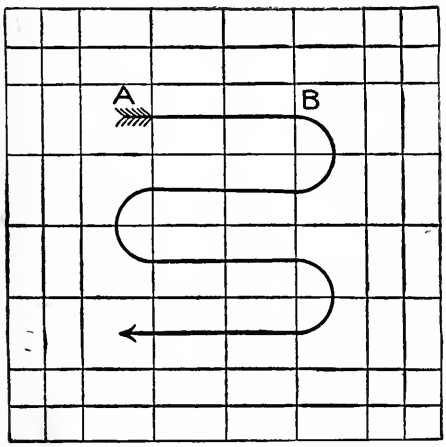


FIG. 96.—Scheme for counting cells overlying ruled surface.

$$\frac{\text{Number of cells counted} \times 4000 \times \text{number of dilutions}}{\text{Number of squares}} = \text{Number of cells per cubic mm.}$$

The greater the number of cells counted, especially with low dilutions, assuming that the mixture is thorough, the more accurate will be the results.

Technic of Counting the Leucocytes.—In determining the number of leucocytes, the red pipette may be used, but more accurate results are obtained with the white pipette, as lower dilutions are secured. A ½ or ⅓ per cent. aqueous solution of acetic acid is employed when using the white counter in order to dissolve the red cells. Except in the case of leukæmic blood, a dilution of 1:20 or 1:10 is most convenient for the majority of leucocytic counts. When the number of white cells is estimated with the red counter, with a 1:100 or 1:200 dilution, Toisson's solution is very useful, since with it the leucocytes are tinted blue and therefore readily distinguished from erythrocytes, which have a yellowish or greenish color. With Zappert's modified ruling the cells overlying a larger area can be counted. The formula for estimating the leucocytes per cubic millimetre is the same as that used for determining the number of erythrocytes. In routine clinical work the corpuscles overlying the entire ruled area of at least 400 squares should be counted when employing dilutions of one in ten or twenty.

Cleansing the Instrument.—After removing the fluid from the pipette, it is rinsed with water, then with alcohol, and finally with ether, and dried thoroughly. An atomizer bulb is useful for expelling the fluid from the tube and for drying. A simple method of removing the fluid from the pipette consists in pressing the end of the rubber tube between the fingers so as to occlude its lumen, and then by twisting the tube the fluid is expelled from the pipette. The counting chamber should be cleaned with water and dried with a soft handkerchief or tissue paper. Alcohol, ether, and xylol should not be used for cleaning the counting chamber, since these substances may dissolve the cement which holds the parts together.

Enumeration of Blood-platelets.—The blood-platelets are rarely seen in fresh unstained specimens, as they disappear almost immediately after the blood is exposed to the air. They are colorless, spherical, oval, or irregular, varying considerably in size, usually from one to three microns. In fresh blood, platelets are demonstrated by placing a cover-glass upon a slide and bringing their edges in contact with the blood as it flows from the puncture. Their number may be approximately estimated by Determan's method as follows: Place a drop of a 9 per cent. aqueous solution of sodium chloride upon the skin and make the puncture through the drop of fluid. As the blood flows from the wound, it is mixed with the reagent by stirring with a cover-glass or slide, and then a part of this mixture is placed upon the Thoma-Zeiss counting chamber and the cover-glass adjusted. The ratio of blood-platelets to erythrocytes is next determined in a given area. The number of red corpuscles per cubic millimetre is found by the Thoma-Zeiss method, and from this figure the actual number of blood-platelets per cubic millimetre can be calculated by the ratio the red cells bear to platelets.

Hæmoglobin Estimation.—The principle involved in the estimation of hæmoglobin with most of the instruments used in clinical work is based upon a comparison of the color of undiluted or diluted blood with a standard color scale.

Dare's Method.—The principle of this method is based on matching the tint of a film of undiluted blood of definite thickness with a graduated color scale. The essential parts of this hæmoglobinometer are a wedge-shaped semicircle of glass stained with Cassius's "golden purple" so that the various depths of the color displayed by the scale represent hæmoglobin

values ranging from 10 per cent. to 120 per cent. (this wedge is contained within a hard-rubber case so that it can be revolved by operating a thumb-screw); a telescoping camera tube supplied with a magnifying lens through which the color of the blood and that of a part of the wedge is viewed; a pipette composed of two plates of glass, one being transparent and the other opaque (white glass); a part of the surface of the latter is slightly bevelled, so that a thin compartment is formed between the plates when their surfaces are opposed; and a candle holder.

Technic.—The pipette is brought in contact with a large drop of blood. It fills by capillarity. The pipette is then placed in its compartment on the side of the case. The light of a candle is used in making the color comparison, the instrument being held in a position so as to avoid direct sunlight. The rapidity with which an accurate hæmoglobin estimation can be made is the greatest advantage of this method. The matching of the colors should be done immediately after filling the pipette, since coagulation may begin within three or four minutes. The tint of the colored wedge of Dare's hæmoglobinometer does not in every instance correspond exactly with the color curve of certain anæmic bloods.

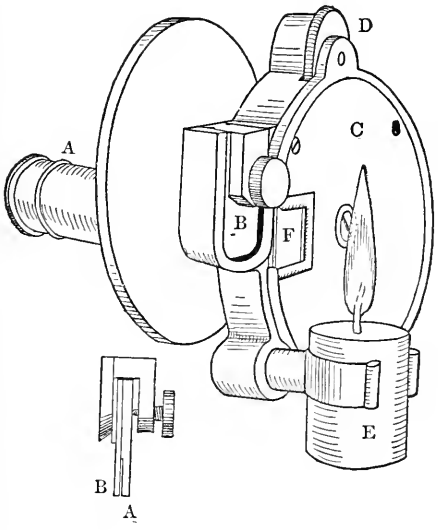


Fig. 97.—1. Dare's hæmoglobinometer. *A*, telescope; *B*, pipette in place; *C*, case enclosing color-prism; *D*, milled head moving prism; *E*, candle; *F*, window admitting light to color-prism. 2. Pipette. *A*, the white glass; *B*, clear glass disk.—Emerson.

Tallqvist's Hæmoglobinometer.—With this method the color of a drop of blood soaked into filter-paper is compared with a color scale lithographed upon paper. The apparatus consists of a book containing sheets of white filter-paper and a lithographed color scale of ten tints representing hæmoglobin values between 10 and 100 per cent.

Technic.—A piece of the white filter-paper is applied to the drop of blood, and, as soon as the moist gloss has disappeared from the surface of the blood-soaked paper, its color is compared with the scale. Accurate results are not claimed for this simple method. An error of at least ten per cent. is unavoidable.

Von Fleischl Hæmometer.—This instrument is composed of the following parts: A metallic stage having a circular opening in its centre, supported by a stand. To the frame of this stand is attached a plaster-of-Paris reflector. A glass wedge, tinted with Cassius's "golden purple," fixed within a metal frame. The depths of the color of the wedge correspond to a scale of hæmoglobin percentages stamped upon the frame, which range from 1 to 120. A cylindrical metallic mixing cell, divided into equal parts by a vertical partition, and provided with a glass bottom. A capillary measuring pipette attached to a metal handle. As the capacity of the

pipettes varies in different instruments, a figure is stamped upon the handle of the pipette and a similar marking on the stage of the instrument for which it is suited. A finely pointed glass dropper, for filling the metallic cell.

Technic.—When one end of the pipette is brought in contact with the blood, secured in the usual manner, it fills automatically by capillarity. Blood adhering to the external surface of the pipette must be wiped away before emptying its contents. After partially filling one of the compartments of the cell with water, the blood is washed out of the pipette with water. The blood and the water are then thoroughly mixed by stirring with the handle of the pipette. The fluid adhering to the handle must then be washed off with water, which is allowed to drain into the mixing compartment. The other division of the cell is filled with water. Avoid moistening the top of the vertical septum, as this may cause the fluids of the compartments to commingle. The filled cell is now adjusted in its proper position on the stage, and a comparison of the color of the diluted blood with that of the scale is made in a darkened room, or with a light-proof box. A candle flame placed about 15 or 20 centimetres in front of the plaster-of-Paris reflector is used for illumination. The operator, standing to one side of the instrument, matches the colors by turning the thumb-screw. The glass wedge should be moved quickly. Never view the colors for more than a few seconds, since the eye is easily fatigued by prolonged inspection. After two readings have been made, the mean of these is taken as the result. An attempt should always be made to compare only the median portion of the color fields, which may be readily accomplished by placing under the glass bottom of the cell a diaphragm of thin metal or paper, having a narrow slit about 4 millimetres in width, the long axis of which is at right angles to the partition of the mixing cell.

When the hæmoglobin percentage is low (below 30), two or three pipettes full of blood should be used, and the result divided by the number of pipettes employed. Degree of error with the von Fleischl instrument is between 5 and 10 per cent.

The Meischer's Hæmoglobinometer.—This modification of the von Fleischl instrument possesses certain advantages over the latter whereby the degree of error is considerably lessened. The principle of Meischer's method is the same as that of von Fleischl. A mixing pipette is employed with which accurate dilutions of 1:200, 1:300, or 1:400 can be secured. For normal blood or nearly so, dilutions of 1:400 are

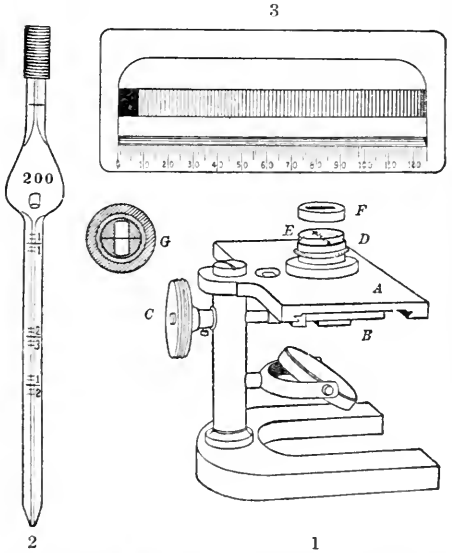


Fig. 98.—1. Meischer's modification of Fleischl's hæmoglobinometer. *A*, stage; *B*, color-prism rack; *C*, milled head; *D*, cell; *E*, cover-glass; *F*, cap; *G*, cell seen from above. 2. Mixing pipette. 3. Color-prism.—Emerson.

most convenient, but with low hæmoglobin values dilutions of 1:200 or 1:300 are more serviceable. Two metallic chambers are employed, each of which is divided by a vertical partition and supplied with a glass bottom. One compartment receives the diluted blood, the other water. One chamber is shallower than the other. The partition dividing the cells is slightly raised so that the glass cover, provided with a groove, may be slid over the top of the cylinder, thereby preventing the fluids from commingling. A lid having a narrow oblong opening is used to cover the chamber so that the width of the field exposed when making the color comparison does not correspond to more than three degrees of the percentage scale. The tinted wedge of this instrument is more accurate than that of the von Fleischl. After securing the desired dilution and mixture in the pipette, one of the compartments in each of the cells is filled with the blood solution, the other compartment with water; the glass cover is then slid into position and the metal top adjusted. The reading of each cell is then made with artificial illumination, using the same technic as with the original von Fleischl method. The result of the reading of the shallower cell is multiplied by $\frac{5}{4}$; this figure should correspond closely with the reading of the other chamber, one result controlling the other. The mean of the two readings represents the hæmoglobin percentage.

Oliver's Hæmoglobinometer.—With this method the color of a definite quantity of diluted blood is compared with a standard color scale, consisting of a series of tinted glass plates. The instrument is composed of the following parts: A standard blood scale composed of 12 colored disks, mounted upon a perfectly white surface in two metal frames. Their tints correspond with the color of various dilutions of blood. These primary disks correspond to hæmoglobin percentages ranging from 10 to 120; two pieces of tinted glass, called riders, are supplied with the instrument for ordinary clinical purposes. When a rider is superimposed upon a primary color, its shade deepens and therefore determines intermediate percentages between those indicated by the disks. An error of $2\frac{1}{2}$ per cent. is unavoidable. A capillary tube having a capacity of 5 cubic millimetres for measuring blood. A standard mixing cell provided with a glass lid. A camera tube through which the colors are viewed, and a pipette for washing the blood out of the measuring pipette.

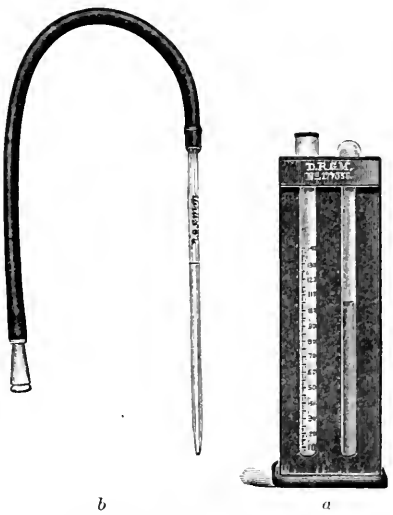
Technic. — The blood measured in the pipette is washed into the mixing cell with water and mixed with the handle of the pipette. The fluid which adheres to the handle is rinsed with the water and the cell filled. The glass lid of the mixing cell is then adjusted in a manner so that a small air bubble is present under the cover. The color of the diluted blood is matched with one of the disks of the color scale in a darkened room, illuminated with the light of a small wax candle placed about 10 centimetres in front of the mixing cell and the color disk. One or both riders may be required to intensify the tint of the primary disk.

Gowers's Hæmoglobinometer.—With this method a definite quantity of blood is diluted, until the color of the mixture corresponds with a standard color contained in a tube. This instrument consists of: A standard color tube which contains glycerin jelly colored with picrocarmine, so that its tint corresponds with that of a solution containing one

part of normal blood in a hundred parts of water; a mixing test-tube having a graduated scale ranging from 5 to 120; a pipette for measuring 20 cubic millimetres of blood.

Technic.—The measuring pipette, to which is attached a small rubber tube, is filled by suction up to the point marked 20. A few drops of water are placed into the mixing tube, then the blood in the pipette is blown into the tube. Water is added in small amounts, shaking after each addition in order to secure a mixture, until the color of the solution corresponds with that of the standard tube. The height of the fluid reached indicates the hæmoglobin percentage. The color comparison is made with daylight by holding the tube against a white background.

Sahli's Hæmometer.—The principle of this method is based on comparing the tint of a standard fluid composed of a definite amount of normal blood and of a decinormal solution of hydrochloric acid with the tint of a solution of blood to be tested treated with a decinormal hydrochloric acid solution and water in sufficient quantity to exactly match the colors. The height of the column of fluid in the mixing tube indicates the hæmoglobin percentage. Sahli claims that with this method the color of the standard solutions and that of the blood properly diluted corresponds quite accurately, thereby insuring uniform results. The apparatus is similar in construction to Gowers's hæmometer. It consists of a sealed tube containing the standard color solution of decinormal hydrochloric acid holding one per cent. of blood; a graduated test-tube for mixing the blood with a decinormal hydrochloric acid solution and water; a pipette for measuring 20 cubic millimetres of blood; a perforated stand with a white glass back for holding the tubes; a bottle for carrying the acid solution; and a finely pointed pipette. The standard color fluid has a brownish-yellow color, due to hæmatin hydrochlorate held in suspension. Since precipitation of this substance will occur on standing, the sealed tube is provided with a glass ball which serves to mix the particles when the tube is agitated.



b *a*

Fig. 99.—*a*, Sahli's hæmometer; *b*, pipette.

Technic.—The graduated tube is filled with decinormal hydrochloric acid to the mark 10. Twenty c.mm. of blood measured in the pipette are then blown into the acid solution and mixed. The measuring pipette is then filled with water and discharged into the mixing tube. The graduated tube is now placed in its compartment in the stand alongside of the standard tube and water is added in small amounts to the blood solution, mixing after each addition, until the color matches the standard tint. The height of the column of fluid in the tube, as indicated by the

graduated scale, represents the hæmoglobin percentage. The test is conducted with natural or artificial light. More accurate readings are possible when the test is made with artificial light in a darkened room.

Color Index.—The terms color index, blood decimal, or blood quotient are used to express the average hæmoglobin richness of the erythrocytes. This factor is determined by dividing the hæmoglobin percentage by the percentage of colored corpuscles per cubic millimetre. The normal color index is expressed by the figure 1, *i.e.*, 100 per cent. of hæmoglobin divided by 100 per cent. of red cells. In anæmic states the same result is obtained when the hæmoglobin and red cells are proportionately reduced. In chlorosis the color index is generally decidedly diminished, while in most symptomatic anæmias it is slightly and in some cases markedly lowered. In pernicious anæmia, except during periods of improvement, it is generally increased.

Estimation of the Relative Volume of Plasma and of Corpuscles.—This determination is made by applying centrifugal force to blood contained in a tube, which separates the corpuscles from the plasma. By estimating the volume of corpuscles, an approximate idea may be formed of the number of cells per cubic millimetre.

Daland's Hæmatocrit.—This instrument consists of a set of gears operating a metal frame into which are fastened two capillary tubes. A hand lever is connected with the gears. The tubes for measuring the blood, graduated into 100 equal divisions, are 50 millimetres in length, with a lumen of ½ millimetre diameter.

Technic.—A piece of rubber tubing with a mouth-piece is attached to one end of the graduated tube. Blood is sucked into the pipette until completely filled. After removing the rubber tubing, the pipettes containing blood are fastened into the metal frame and immediately the handle of the instrument is turned for 3 minutes, at the rate of about 77 revolutions per minute, which produces the speed desired. The centrifugal force separates the blood into three layers; the one most distant, of dark red color, is composed of erythrocytes, the middle one, of milky color, is formed of leucocytes, while the inner clear layer consists of plasma. With normal blood the column of erythrocytes reaches to the graduation marked 50 or 51; each division of the scale approximately represents 100,000 corpuscles per cubic millimetre. Accurate estimations of the number of cells per cubic millimetre is impossible, since the size of the erythrocytes varies in pathological conditions and because a uniform speed is almost impossible to obtain. Variations in the centrifugal force will produce differences in the degree of compactness of the cells. The number of leucocytes can only be roughly estimated when there is a marked increase, as in leukæmia, but under normal conditions or pathological states with slight or moderate variations the leucocytic layer is too indistinct to warrant an opinion as to their number. The pipettes of this instrument should be cleaned immediately after using by passing a fine wire through the lumen, then washing with water, followed by alcohol and ether.

Volume Index.—Volume index, the term applied to represent the average volume of the erythrocyte, is determined by dividing the per-

centage volume, as estimated with the hæmatocrit, by the percentage of the erythrocytes per cubic millimetre, obtained with the hæmocytometer.

Estimation of Specific Gravity.—An accurate estimation of the specific gravity of the blood can be obtained by Schmaltz's method, which consists of weighing a dry pipette upon a sensitive balance. The pipette is then filled with water and the weight determined. After cleaning and drying, the pipette is filled with blood and again weighed. From these figures the specific gravity is calculated.

Hammerschlag's Method.—Hammerschlag's modification of Roy's method is based upon the principle of suspending a drop of blood in a liquid having the same specific gravity. The specific gravity of the suspension fluid is then determined with a hydrometer, which corresponds to that of the blood.

Technic.—Pour benzol and chloroform into an hydrometer jar, in such proportions as to secure a mixture having a specific gravity of about 1.060. Partially fill a pipette, or medicine dropper, with blood and insert it into the benzol-chloroform solution; expel a droplet into the fluid. If the blood is lighter than the mixture, it will rise to the top. Benzol should then be added and the fluid carefully stirred with a glass rod until the blood is suspended in the mixture. The specific gravity of the benzol-chloroform solution is next determined, which corresponds to that of the blood. If the specific gravity of the blood is greater than that of the benzol-chloroform mixture, causing the blood to sink, the addition of chloroform is necessary to cause suspension. This method of determining the specific gravity is seldom employed in clinical work, as it is tedious and as errors of technic are readily made. The specific gravity ranges of the blood correspond quite closely to definite hæmoglobin percentages; notable exceptions to this rule are found in progressive pernicious anæmia, where the hæmoglobin percentage is slightly higher than the specific gravity indicates, while in leukæmia the reverse is observed. Hammerschlag's scale of specific gravity ranges with equivalent hæmoglobin percentages is as follows:

Spec. Gravity.	Hæmoglobin.
1.033–1.035	25–30 per cent.
1.035–1.038	30–35 per cent.
1.038–1.040	35–40 per cent.
1.040–1.045	40–45 per cent.
1.045–1.048	45–55 per cent.
1.048–1.050	55–65 per cent.
1.050–1.053	65–70 per cent.
1.053–1.055	70–75 per cent.
1.055–1.057	75–85 per cent.
1.057–1.060	85–95 per cent.

Estimation of the Time of Coagulation.—As a number of conditions influence the rapidity with which coagulation of the blood occurs after it is withdrawn from the blood-vessels, such as the amount of blood and the temperature, the results obtained by different methods of determining the clotting time are not available for comparative studies. In this connection it should also be borne in mind that the factors which control intra- and extravascular coagulation are in all likelihood dissimilar.

Method of Russell and Brodie.—The coagulation time is determined by microscopical study of the blood. The apparatus needed for this method is provided with a moist chamber having a glass bottom. A removable glass cone (the lower surface of which is 4 mm. in diameter) forms the upper portion of the chamber. A current of air is introduced into the chamber by means of a small tube one end of which projects into the cell, while to the other end is attached a rubber tube supplied with a bulb. Boggs's coagulometer, a modification of the instrument just described, is equipped with an improved glass cone and a metal tube.

Technic.—A drop of blood is placed upon the lower surface of the cone which is then immediately fitted into the chamber. The instrument is then put upon the stage of the microscope and with a low-power objective the blood is brought into focus. At successive intervals the blood is agitated by means of the current of air sent into the cell from the bulb. It will be noted that at first the stream of air causes the corpuscles to move freely. A little later clumps form in the peripheral zone of the blood and these can be advanced by the air current. Then as clotting progresses masses of blood-cells cease to move freely, the drop alters its shape, and the corpuscles exhibit a concentric motion. Lastly, a radial movement appears, clumps of cells being displaced by the air current towards the centre and these quickly return to their original position. Clotting is now considered complete. The normal coagulation time as determined by this method varies from three to eight minutes, the average time being about five minutes.

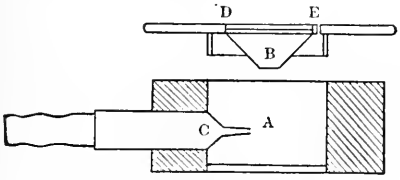


Fig. 100.—Coagulometer of Russell and Brodie as modified by Boggs. *A*, moist chamber; *B*, cone of glass, the lower surface of which holds the drop of blood; *C*, side tube; *D* and *E*, cover-glass; at *E*, a pinhole.—Emerson.

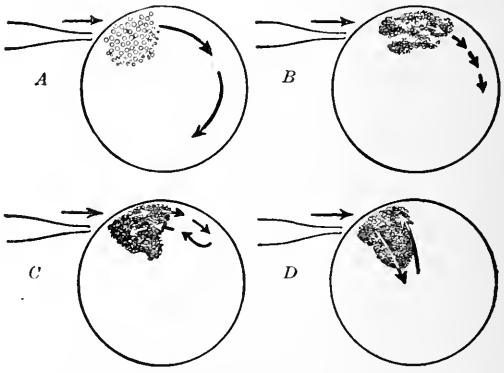


Fig. 101.—Diagram to illustrate the movement of the cells during coagulation.—Emerson.

Wright's Method.—The coagulometer devised by Wright consists of a cylindrical tin vessel provided with a perforated partition, the openings of which are so arranged as to support twelve graduated tubes and a thermometer. The tubes are graduated for 5 c.c. of blood, and are numbered from one to twelve.

Technic.—Water having a temperature of 18.5° C. is poured into the metal container. The blunt end of six or eight of the tubes is then covered with a rubber cap. The tubes are then placed, closed end downward, into the water. After having acquired the temperature of the water, they are removed separately at once or one-half minute intervals, the cap taken off, filled with 5 c.c. of blood and immediately replaced into the water without reapplying the caps. Attempts

at short intervals are made to dislodge the blood from the tubes by blowing. When the blood cannot be removed from one of the tubes, coagulation may be considered complete. The clotting time is the difference of time between the filling the tube and the unsuccessful attempt to expel its contents. With this instrument the coagulation time of normal blood in most instances is from three to six minutes, although the period may be as long as fifteen minutes.

Bacteriological Examination.—There are two methods of demonstrating bacteria in the blood—one by an immediate miscroscopical examination of stained films, the other by blood culturing. The former plan is applicable only in a limited number of diseases and cannot be employed as a routine procedure because the blood obtained by puncturing the skin frequently becomes contaminated with bacteria which are normally present in the skin, especially the staphylococcus epidermidis albus. However, this method has been found convenient in the diagnosis of very severe cases of bubonic plague. Films can be prepared by placing several large drops of blood upon a slide so as to secure a thick spread. Before staining, much of the hemoglobin may be removed from the dried film by soaking the specimen in distilled water. Far more reliable results are obtained by removing 5–10 c.c. of blood from a vein and diluting it with 100 c.c. of bouillon. From this mixture the bacteriological investigations are then made. In the late stages of anthrax infection the blood may show a large number of bacilli so that the immediate microscopical method may give positive results. In this disease the cultural method gives more certain results than the study of films prepared from blood obtained by puncture of the skin.

Technic.—The skin of the flexor surface of the elbow is cleansed as for a surgical operation, by scrubbing thoroughly with soap and water, washing with sterilized water, alcohol, and ether, after which an antiseptic dressing is applied and allowed to remain for six or eight hours. The operator, having prepared his hands, should, after removing the antiseptic dressing, wash the skin with sterilized water. A syringe (of moderate size like the instrument used for exploratory puncture) or a special "blood aspirator" is required to remove the blood from the vein. A most useful instrument employed by many workers consists of a graduated glass tube having a capacity of about 10 cubic centimetres, one end of which is fitted to a No. 42 hypodermic needle, and into the other end a small plug of cotton is inserted. In order to sterilize the instrument, it is placed in a large glass tube, the ends of which are then plugged with cotton. After sterilization a piece of rubber tubing is fastened to the end of the aspirator containing the cotton. A bandage is wound around the arm of the patient so as to obstruct the venous circulation, and when the superficial veins at the elbow become distended, the needle of the syringe or blood aspirator is inserted into the most prominent vessel. The piston is withdrawn slowly until the desired quantity of blood is obtained. As a rule, the blood flows freely into the aspirator previously described, but, should this not be the case, a sufficient amount can be secured by making suction through the rubber tube.

The blood is placed in a suitable culture medium. Fluid media such as bouillon and litmus milk are generally selected for the primary inoculations when certain types of bacteria are suspected, while agar may be chosen when the medium is to be plated. One or two cubic centimetres of blood are added to 50 or 100 c.c. of fluid medium so that dilutions of one in fifty or one in one hundred are secured. For details of bacteriological technic, which do not fall within the scope of this work, special treatises on bacteriology should be consulted.

Fig. 102.—Capillary pipette.

Agglutination Reaction.—The blood in certain stages of typhoid fever, and often after the attack, possesses the property of checking the motility of typhoid bacilli and causing these organisms to form into clumps. This agglutination phenomenon is so pronounced that high dilutions of blood, as one in fifty or one hundred, or even higher, give positive results. The blood in similar dilutions in other diseases and in health does not act in this manner with typhoid bacilli. With low dilutions, however, a positive agglutination reaction is often present with normal or abnormal blood. In a number of diseases—as pneumococcal and streptococcal infections, paratyphoid fever, Malta fever, tuberculosis, cholera, plague, relapsing fever, glanders, and others—specific agglutination reactions have been obtained. The agglutination test is chiefly employed in the diagnosis of typhoid and paratyphoid fevers and is generally spoken of as the Widal, Gruber-Widal, or Pfeiffer-Widal reaction. For this test two methods are available, (1) the microscopic and (2) the macroscopic. In typhoid fever the agglutination reaction is positive in about 97 per cent. of the cases during the course of the disease, manifesting itself in a majority of them about the end of the first or during the second week, in a few instances as early as the third or fourth day, while in others it is not obtained until the attack is far advanced, and it often persists long after convalescence. The intensity of the reaction varies in different cases. Positive reactions are obtained with dilutions as high as 1:200. In some instances the reactions occur almost instantly, while in others the stoppage of motility and clumping take place slowly.

1. **Microscopical Serum Test.** — This test may be performed with fluid blood, blood-serum, or dried blood.

Technic.—*Preparing Cultures.*—From a slant agar growth of typhoid bacilli, preferably not older than one month, subcultures are made in sterile bouillon and incubated at blood heat for 8 to 12 hours, when they are ready for use. The stock culture should be kept in a cool place. Some workers prefer a suspension of typhoid bacilli in salt solution made by placing a loopful of a twenty-four hour agar growth in saline solution. The tube containing the fluid is agitated until a uniform suspension of the germs is obtained.

Collecting and Diluting Serum. — A capillary pipette, suitable for measuring the blood, is made from a piece of glass tubing about 30 cm.

in length and 5 or 6 mm. in diameter. The middle portion of this tube is heated in the Bunsen flame, rotating continuously in its long axis until the glass is thoroughly softened over 3 to 6 centimetres of its length; remove from the flame and draw the two ends apart with a steady uniform pull so that the heated portion tapers into a long capillary tube. By melting the middle of the capillary tube in the flame, two pipettes with the capillary end sealed off are made. A Wright's blood capsule, shown in Fig. 103, will be found convenient for collecting the blood.

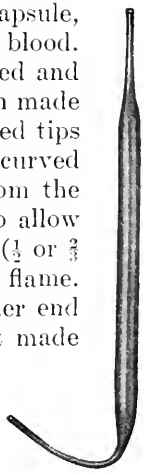


Fig. 103.—Tube for serum work.—Emerson.

Preparing the Serum.—The patient's finger-tip is cleansed and rubbed briskly so as to produce hyperæmia. A puncture is then made of sufficient size so as to insure a good flow of blood. The sealed tips of a Wright's capsule are broken off, and the end of the short curved portion of the capsule is placed into the blood as it issues from the small wound, the body of the tube slanting downward so as to allow the blood to enter by gravity. The capsule is partly filled ($\frac{1}{2}$ or $\frac{2}{3}$ full). The tip of the longer arm is sealed off by heating in a flame. When properly cooled, the blood is shaken down. The other end may then be closed to prevent evaporation, if the test is not made immediately. The capsule is now hooked upon the rim of a centrifuge tube and centrifugalized until clear serum separates. Slight turbidity of the serum does not interfere with the test. The capsule containing the centrifugalized blood is opened by filing a groove into the glass tube above the level of the serum and breaking off the end. The fine end of a capillary pipette (having previously been broken off the sealed tip) is inserted into the capsule and the serum drawn into the tube. The blood-serum may be diluted and mixed with the culture in watch crystals or in a porcelain plate having a number of cup-shaped depressions as shown in Fig. 104. One drop of serum is now placed into one of the depressions of the porcelain plate. Dilutions of the serum with sterilized normal salt solution are then made. The capillary tube, having been cleaned with salt solution or water, is partly filled with saline fluid. Into the depressions containing a drop of serum, 24 drops of salt solution are allowed to fall from the pipette and mixed, thus making a dilution of 1–25, since the drops from the pipette are practically of the same size. Into a second depression are placed 5 drops of salt solution and 5 drops of the diluted serum of 1–25, securing a 1–50 dilution. More accurate results are obtained by measuring and diluting the serum in a Thoma-Zeiss hæmocytometer pipette. Two hanging drop preparations are prepared—one from each dilution—by mixing upon a cover-glass a platinum loopful of bouillon culture of typhoid bacilli with a loopful of diluted serum. Since each dilution of serum is again diluted to $\frac{1}{2}$, the proportions now stand 1–50 and 1–100. The cover-glasses are adjusted upon the slides and the edges of the slips surrounded by petrolatum to prevent evaporation. The

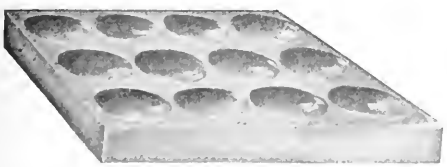


Fig. 104.—Porcelain disk for mixing serum, salt solution, and bacterial cultures.

preparations are allowed to stand at room temperature for exactly one hour. In order to secure correct results, it is essential that the motility of the bacilli should be active, and the density of the culture be uniform and not show clump-like gatherings.

Recording Results.—At the end of one hour, the hanging drop slides are examined microscopically. When motion of the bacilli is found absent and clumping good in both slides, the reaction is termed "positive," but when the free motion without clumping of bacteria is noted, the test is negative. Variations between these two extremes may be recorded according to the judgment of the examiner. Thus, if 1–50 shows no motion and good clumping, but 1–100 exhibits slight motion and only fair clumping, the reaction may be called "very suggestive;" or, again, if 1–50 shows slight motion and poor clumping, while 1–100 free motion and no

FIG. 105. — Widal test. Field of motile organisms.—Emerson.

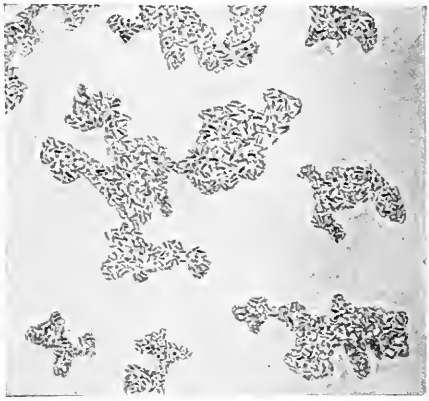


FIG. 106. — Widal test. Field of agglutinated organisms.—Emerson.

clumping, the reaction may be called "slightly suggestive." It is best to indicate definitely the results of each dilution, as for example:

1–50 Good clumping.	No motion.
1–100 Fair clumping.	Slight motion.

This allows the diagnostician to form his own conclusions and does away with dogmatic assertions, such as "Widal positive" or "Widal negative," which are so often a matter of personal equation upon the part of the laboratory worker.

The liquid serum method, unfortunately, cannot always be employed by physicians in active practice. The microscopical test may be carried out with blood collected upon a piece of paper, or upon a slide and allowed to dry, after which the test may be made at any time. The blood secured in this manner is moistened and dissolved in sterilized water, and then diluted and mixed with the culture in the desired proportions. It is obvious that accurate dilutions are impossible, an objection to this method.

2. **Macroscopical Serum Test.** — By aspirating a vein, a sufficient amount of blood is collected in a sterile test-tube and allowed to clot, so as to separate the serum, or the blood may be centrifugated. The

serum is mixed with salt solution and bacterial culture in the desired proportions (1–50 or 1–100). In the case of a positive reaction a flaky precipitate will separate with a clear supernatant fluid, while a negative reaction shows uniform turbidity of the fluid. The macroscopical test may also be performed by mixing bouillon and serum in proper dilutions and inoculating the mixture with a loopful of a broth culture. The presence of a precipitate in the tube at the end of twenty-four hours' incubation signifies a positive result. The chief objection to the macroscopic method is the relatively large amount of blood required. The microscopic test is generally employed in clinical work. The macroscopical test may also be performed with dead cultures of the bacilli. The principle of the Ficker "Typhus Diagnosticum" is based on mixing a dead culture with diluted blood-serum. Bacilli in liquid media killed with carbolic acid or formalin are also employed for this test.

Opsonic Index of the Blood and Its Determination. — Leishman in 1902 devised a method for estimating the phagocytic activity of the leucocytes. Extensive researches upon this subject have recently been made by Wright and Douglas, and many other investigators. Opsonins are substances within the blood which prepare bacteria for ingestion by the white cells. The power of the leucocytes alone to ingest bacteria, the so-called "spontaneous" phagocytosis, has been shown to be very slight, and the rôle played by them in fighting diseases is merely as scavenger, collecting bacteria acted upon by the opsonins. Opsonins do not stimulate or otherwise affect the leucocytes. These substances are destroyed by a temperature of 65° C. for ten minutes.

Technic. — The special technic used for the determination of the opsonic power of the blood may be briefly set forth as follows: There must be on hand for the test (1) an emulsion of the bacteria in salt solution, (2) washed white blood-cells, taken from any source, (3) serum from the patient's blood, and (4) serum of normal blood or from a mixture of healthy bloods taken as a standard control.

Preparing the Bacterial Emulsion.—The micro-organisms for the test are inoculated upon culture medium. For some forms of bacteria, as the Staphylococcus aureus, an agar medium is selected. After twenty-four hours of incubation at 37° C. a fair-sized colony, found on the culture medium, is removed and mixed with a sterile .85 per cent. salt solution. The resulting bacterial emulsion is drawn up and down in a small pipette by means of rubber teat. The emulsion is set aside for a few minutes so as to allow the bacterial clumps to settle. The supernatant liquid is then removed and diluted to the desired density. Centrifugalizing may be necessary to separate bacterial clumps. With bacteria not readily emulsified, such as tubercle bacilli, grinding between glass plates or in an agate mortar is required to disintegrate the masses. The emulsion in case of tubercle bacilli may be made from fresh cultures or from dry, dead germs, such as are obtained in the production of tuberculin. Tubercle bacilli are best emulsified with a 1.5 per cent. salt solution. The proper density is one which on mixture with a normal serum and with the leucocytes in equal proportions will show that after incubation an average of 5 or 6 germs have been phagocytozed by each leucocyte. The density of the standard

bacterial mixture may be fixed by McFarland's nephelometer, or by counting the bacteria in a given amount of emulsion in a Thoma-Zeiss counting chamber.

Obtaining the Washed Leucocytes.—A test-tube is filled two-thirds full with an aqueous solution containing 1.5 per cent. sodium citrate and .85 per cent. sodium chloride. The finger is pricked and 8 to 12 drops of blood are allowed to fall into the tube. The solution is shaken and then the tube is placed in an electric centrifuge and centrifugalized for 5 minutes at a speed of 1500 to 2000 revolutions per minute. The citrate defibrinates the blood and prevents clotting, while the sodium chloride solution washes the cells free of serum. Upon removal from the centrifuge, the tube is found to contain a compact sediment of blood-cells with a clear or very slightly cloudy supernatant fluid which consists of serum and salt solution. Overlying the surface of the red sediment will be found a white coating termed the "creamy layer," which is formed principally of white cells. The clear supernatant fluid is now drawn off with a capillary pipette by means of a rubber bulb. The layer of leucocytes, which contains some red cells, is now carefully removed from the compact layer of erythrocytes with the capillary pipette and placed in a small glass tube having a sealed end. The leucocytes may be washed with saline solution several times in order to remove the sodium citrate. This is accomplished by placing the leucocytes in a centrifuge tube or small test-tube and partly filling it with .85 per cent. salt solution. The tube is then centrifugalized, after which the supernatant fluid is removed with a pipette. This operation may be repeated.

Obtaining the Serum.—A Wright's capsule is filled two-thirds full of blood, obtained from the patient, and centrifugalized until the serum is clear. Serum must also be obtained from normal blood. Having on hand the bacterial emulsion, washed corpuscles, and serum, the main part of the test may be carried out. By means of a capillary pipette, equal amounts of bacterial emulsion, white blood-cells, and serum are measured, and the fluid mixed on a slide or watch crystal by drawing the material up and forcing it down the capillary pipette. The fluids are measured in the following manner: The rubber teat attached to the pipette is compressed, and, by gently relaxing the pressure, white cells, bacterial emulsion, and serum, in the order named, are drawn into the capillary bore up to the mark indicated by the pencil mark, each column being separated, by a small air bubble. Two pipettes are necessary for one test, one for the patient's serum, the other for the control serum. A special pipette, supplied with a rubber teat, is often used to measure and mix the bacterial emulsion serum and washed leucocytes, and is constructed in such a manner as to allow the worker a means of controlling accurately the amount of fluids drawn up in the long arm of the pipette and of mixing the contents afterwards. The sealed tip of a capillary pipette having been broken off squarely, a pencil mark is made 2 or 3 cm. above its extremity. After the ingredients are thoroughly mixed, the fluid is drawn into the pipette and its end sealed in the flame. The tube is then placed in the incubator at 37° C. for 15 minutes. In a like manner, the control test is prepared with equal amounts of white corpuscles, bacterial

emulsion, and normal serum, which are also incubated. After incubation the end of the pipette is broken off and the contents are run up and down so as to mix thoroughly. Smear preparations are made of the material from each pipette upon slides or cover-glasses. After fixation the smears are treated with any reliable stain, such as Leishman's, which brings out distinctly the leucocytes and bacteria. For tubercle bacilli, carbol fuchsin and Gabbett's or Pappenheim's stain may be employed. The specimens are now examined with an oil-immersion lens. The number of bacteria in 100 typical polymorphonuclear neutrophiles is determined in both specimens. The average number of bacteria per leucocyte is then calculated for each specimen, which constitutes the phagocytic index. The phagocytic index of the patient's serum divided by the phagocytic index of the normal or control serum gives the opsonic index. The test can only be carried out properly in a well-equipped laboratory by one who has mastered opsonic technic. The strength of the bacterial emulsion, the length of incubation, the age of the ingredients employed, and the personal equation are some of the factors which influence the results. Opsonic index is employed in the diagnosis and prognosis of certain infectious diseases and in gauging the dose and the frequency of administration of bacterial vaccines. After the injection of therapeutic doses of bacterial vaccines, the index is seen primarily to fall and soon afterwards to rise above the normal. The initial fall constitutes the "negative phase" and the rise the "positive phase." The vaccine should not be repeated until the negative phase has passed into the positive, and this can only be gauged by repeated observations of the index. The value of the opsonic index for therapeutic or diagnostic purposes has not been definitely settled.

Test for the Detection of Glucose in the Blood.—The presence of hyperglycæmia is so important clinically that the amount of sugar in the blood must be determined exactly. The Lewis-Benedict method gives a very accurate estimation. The red color secured by heating a glucose solution with picric acid and sodium carbonate is used as a basis of the colorimetric determination.

Technic.—Dilute 2 c.c. of blood with 8 c.c. of water and add 15 c.c. of a concentrated picric acid solution. The proteins are precipitated. Filter; add 2 c.c. of concentrated picric acid solution and 1 c.c. of 10 per cent. sodium carbonate to 8 c.c. of the filtrate. Evaporate to a small volume or to dryness, add a small amount of water and bring to the boiling point. Transfer to a 10 c.c. flask and make up the volume by addition of water, filter and compare in a Duboscq colorimeter with a standard pieramic acid solution that is so prepared as to correspond in color to that produced by 0.64 mg. of dextrose under conditions described in test. According to these authors the blood-sugar content of healthy persons averages about 0.1 per cent.

Test for the Determination of Acetone in the Blood.—Dilute 10 c.c. of blood with 40 c.c. of a .5 per cent. solution of potassium oxalate and distil 15 c.c. from this mixture. Acidulate with H_2SO_4 and re-distil 5–8 c.c. The acetone is in the second distillate. Determine its presence and quantity by the iodoform method of estimation.

General Results of Blood Examinations.

Volume. — The blood, which forms from 4 per cent. to 7 per cent. of the total body weight, is a highly specialized tissue, consisting of erythrocytes, leucocytes, blood-plaques, and hæmokonia, suspended in a liquid matrix, the plasma. In health the total volume of blood varies within narrow limits. The view formerly entertained that an increase in the total amount is constantly present in some individuals—*plethora*—is not sustained by recent researches. *Oligœmia,* or a decrease in the total quantity,—e.g., due to a copious hemorrhage,—persists only for a short time after the bleeding, as the volume is rapidly brought up to its normal standard by the absorption of fluid from other tissues, which dilutes the remaining blood, producing a condition termed *hydræmia* or *serous plethora.* Rapid abstraction of watery elements from the blood by sweating, diarrhœa, or vomiting causes a transitory increase in its density, known as *anhydræmia.*

Color.—The color of the arterial blood is bright red, due to the presence of a large amount of oxyhæmoglobin, while that of venous blood, which contains less oxyhæmoglobin and much carbon dioxide, is dark red or purple. In some pathological states, as in diabetes mellitus and in leukæmia, the blood often has a milky tint; a peculiar chocolate color is sometimes imparted to the blood by poisoning with potassium chlorate, nitrobenzol, and hydrocyanic acid. Imperfect aëration, encountered in some diseases of the respiratory organs and heart and in chronic polycythæmia with splenic enlargement (Osler's disease), causes dark red blood similar to the color of venous blood. In carbon monoxide poisoning it is bright scarlet.

Reaction.—The reaction of normal blood is alkaline. The degree of alkalescence varies considerably both in health and in disease. None of the methods of determining the intensity of this reaction has been generally adopted for routine clinical purposes, and, as the results of various methods are not uniform, comparative studies by different observers are in the main inaccurate. The adoption of some standard technic may establish definite results, but up to the present time the data bearing upon this subject are insufficient to warrant positive opinions. Statistics indicate that the alkalinity is lowered in many pathological conditions, notably in diabetic coma, in many of the infectious diseases, especially in Asiatic cholera, in organic hepatic disease, in uræmia, in cachectic states, in a considerable group of skin affections, in poisoning by mineral acids, and in a number of other conditions. In chlorosis and rheumatic fever it has been found increased.

Specific Gravity.—The specific gravity of normal blood is about 1.060. It fluctuates slightly in health, while in disease there are wide oscillations. The specific gravity range is decidedly influenced by the amount of hæmoglobin, and so close is the relation between the two that an approximate hæmoglobin estimation can be made by determining its specific gravity. Exceptions to this rule are found in the case of leukæmia, in which the range of specific gravity would indicate a higher hæmoglobin value than actually exists, while in pernicious anæmia the reverse is true.

Coagulation of the Blood. — Within a short time after blood has been withdrawn from the circulation of a healthy individual, it undergoes coagulation, a process which determines the formation of fibrin and the separation of a clear, straw-colored fluid, the blood-serum. In a number of diseases considerable importance is attached to the determination of the time required for clotting. Delayed coagulation is encountered in persons suffering from obstructive jaundice, purpura, scurvy, and hæmophilia. In pernicious anæmia, in some cases of leukæmia, and at times in Hodgkin's disease, the rate of coagulation is prolonged. In some of the infectious fevers, and in acute inflammation attended with abscess formation, clotting is retarded, while in chlorosis, pneumonia, and scarlet fever it is rapid.

The Plasma.—The plasma, a complex albuminous body, which holds in suspension the solid elements and in solution many organic and inorganic compounds, is the vehicle through which substances are transported to the tissues and waste products carried to the excretory organs. The plasma also holds certain bodies possessing antitoxic, bactericidal, agglutinative, and opsonic properties. Agglutinins are of importance in the diagnosis of many infectious diseases, as in enteric fever, paratyphoid infections, Malta fever, cholera, relapsing fever, and dysentery. Some observers have noted this reaction in tuberculosis, pneumococcus and streptococcus infections, plague, and leprosy.

Erythrocytes. — The red blood-corpuscles in a preparation of fresh blood, taken from the peripheral circulation of a healthy person, appear as pale yellowish-green, non-nucleated, flattened, biconcave cells of a circular outline. They are pliable, somewhat elastic, non-amœboid, transparent, and show a tendency to form into groups or rolls when withdrawn from the circulation, and consist of a fine stroma which holds an albuminous iron compound, the hæmoglobin. Structural alterations of these cells occur when blood is removed from the circulation. They occasionally exhibit amœboid activity and may undergo disintegration, fragmentation, vacuolation, and crenation. A crenated corpuscle is a shrunken cell from which knob-like processes project. Structural alterations similar to those caused by withdrawing the blood from the vessels occur within the circulation as a result of pathological factors.

Staining Reaction of the Erythrocyte. — The normal red blood-cell, when properly fixed, has a monochromatophilic reaction, showing a selective affinity for acid dyes, while the living cell does not absorb stains (achromatophilic). On account of its biconcavity, the central part of the cell stains less intensely than the peripheral zone. The long diameter of the majority of the healthy cells measures about 7.5 microns, while its variations are between 6 and 9 microns.

Hæmogenesis and Hæmolysis.—It appears to be definitely established that in the adult the red bone-marrow is the chief, if not the only seat of erythrocytic formation. The colored cells develop from nucleated elements, erythroblasts, situated along the walls of capillary spaces of the marrow. Some authorities contend that erythroblasts and certain forms of immature leucocytes are derived from a common ancestral cell. The spleen and lymphatic glands are regarded by some as sources of

erythrocytic formation, a view which is not entertained by many writers. The fairly uniform number maintained in the circulating blood of the healthy individual depends upon the existence of a parallelism between the rate of formation and the rate of destruction. Pathological erythrocytic destruction, unless excessive or prolonged, excites augmentation in the activity of erythroblastic multiplication and is followed by an increase in the output of red cells from the marrow. Most authorities maintain that the liver and in a less degree the spleen and the gastro-intestinal capillary area are concerned in destroying weakened, degenerated, or necrotic cells, while some hold that the bone-marrow also has a hæmolytic function. The coloring material derived from the disintegrated cells is in part transformed, in the liver, into bile pigment and eliminated through the biliary channels, in part discharged by the kidneys, and probably, to a considerable extent, stored up in many of the tissues where it is available for future needs of the body.

Number of Red Blood-cells. — The normal number of erythrocytes, which is 5,000,000 per cubic millimetre for an adult male and 4,500,000 for an adult female, is subject to slight variations under certain physiological conditions and to pronounced alterations in many morbid states. A decrease in the number is termed *oligocythæmia*, while an increase is designated *polycythæmia*. High counts are at times an indication of a decrease in the volume of plasma causing a relative polycythæmia. After blood transfusion, and after active blood regeneration, a temporary rise may be noted. Polycythæmia is encountered in the new-born, where it exists for some days after birth—probably not exceeding ten—in individuals residing in high altitudes, and in robust and well-developed persons. Massage, electricity, and cold bathing may also induce an increase in the erythrocytes in the peripheral blood. A slight reduction in the erythrocytes is brought about by pregnancy, menstruation, and lactation; it is also met with in poorly nourished individuals, in those who are fatigued, and during the period of digestion. Oligocythæmia, due to physiological causes, may sometimes be accounted for by temporary dilution of the blood, while in other instances an absolute decrease in the number of cells offers the best explanation.

In pathological states a relative transitory increase arises when the output of fluid from the body is decidedly in excess of the intake, and is therefore conspicuous in diseases associated with marked polyuria, as diabetes, with copious sweating, as from night sweats of pulmonary tuberculosis, with frequent vomiting, with profuse diarrhœa, as in Asiatic cholera, and after the withdrawal of a large quantity of fluid from a serous cavity, which rapidly reaccumulates, thereby draining the blood of much fluid. The pathological factors responsible for oligocythæmia are numerous, and in the vast majority of diseases associated with lowered counts the reduction depends upon increased blood destruction, in some it may be due to defective blood formation, or to a combination of both of these factors, while in others a slight transitory decrease is brought about by blood dilution—whenever the amount of fluid taken into the body is above the output of liquid. Lowered erythrocytic standards are noted in the primary anæmias, notably pernicious anæmia, in which the figure

often falls as low as one million, and in occasional instances below half a million. Secondary anæmias arise from a great variety of causes, as from infections due to bacteria and animal parasites, metallic poisoning, organic visceral disease, hemorrhage, and many others.

The Hæmoglobin. — Hæmoglobin, a complex albuminous compound containing iron which is a component of the red cells, normally exists in two chemical states, in combination with oxygen (oxyhæmoglobin) and as reduced or plain hæmoglobin. Normal blood contains about 14 per cent. of hæmoglobin. A reduction of hæmoglobin, termed *oligochromæmia*, is generally associated with a fall in the number of erythrocytes. This deficiency of corpuscles, as a rule, is not so marked as the hæmoglobin loss, although sometimes the number of colored elements remains normal; rarely there is a proportionate reduction of the coloring matter and of the number of cells, or the latter may even show a greater percentage reduction than the hæmoglobin. An insufficient amount of hæmoglobin in the corpuscles is brought about by an effort on the part of the bone-marrow to rapidly regenerate cells at the expense of perfect formation (*i.e.*, after or during rapid hæmolysis). A high color index occurs in pernicious anæmia. Investigations, both experimental and clinical, have demonstrated that the hæmoglobin rises in certain anæmic states by the administration of iron compounds.

Hæmoglobinæmia, the term which implies the presence of hæmoglobin in solution in the plasma, is due to a number of causes, and is at times followed by the excretion of hæmoglobin by the kidneys (hæmoglobinuria). The disease known as paroxysmal hæmoglobinuria is a striking example of the latter condition. Among the conditions capable of producing hæmoglobinæmia are poisoning by sulphuric acid, nitrobenzol, phenacetin, acetanilid, phenol, hydrochloric acid, potassium chlorate, mushrooms, and snake venoms. Hæmoglobinæmia is excited by some of the infectious diseases, as malarial fever, enteric fever, yellow fever, typhus fever, variola, septicæmia, diphtheria, and syphilis; also by malignant jaundice, scurvy, sunstroke, burns, and from exposure to intense cold.

Methæmoglobin, another combination of oxygen and hæmoglobin, is a component of some pathological bloods and is present in poisoning with such substances as potassium chlorate, aniline, amyl nitrite, potassium permanganate, antifebrin, nitro-benzol, hydrochinone, potassium ferrocyanide, and snake venom. Carbon monoxide hæmoglobine, which occurs in coal-gas poisoning, gives the blood a bright scarlet appearance.

Abnormal Erythrocytes. — Cells exhibiting abnormal variations in size are common in anæmic states, particularly small erythrocytes known as *microcytes*, which have a diameter of less than 6 microns, and when these forms predominate in the blood the condition is termed *microcytosis* or *microcythæmia*. This change is conspicuous in chlorosis and in some secondary anæmias of the chlorotic type. Minute erythrocytes, spherical in form, with a deeply colored protoplasm, are often described as Eichhorst's corpuscles. Cells measuring more than 9 microns are designated *macrocytes*, and when these abnormally large forms outnumber other colored elements *macrocytosis* or *macrocythæmia* exists. Typical cases of pernicious anæmia show an average increase in the size of the red cells.

Rapid or defective blood formation has been advanced as the factor responsible for microcytosis, although structural alteration in the cells after they have entered the circulation, such as fragmentation and loss of hæmoglobin, may account for some of these dwarfed elements. Macrocytosis appears to depend upon the development of large cells in the marrow, or perhaps it is due to swelling of the protoplasm of the erythrocytes while in the general circulation.

Poikilocytes are cells having an irregular or distorted outline and often appear as pear-shaped, elongated, oval, and "hour-glass" forms. These pathological cells show wide variations in size and in staining peculiarities. The degree of cell deformity and the extent of the variation in size are generally proportionate to the severity of the anæmia. Pernicious anæmia and grave secondary anæmias reveal poikilocytosis in its most typical form. Cells so deficient in hæmoglobin that a mere colorless shell remains are termed phantom or shadow corpuscles or achromacytes. Erythrocytes which react indifferently and irregularly to acid and basic dyes, staining diffusely with both,—termed *polychromatophilic cells,*—are observed in profound anæmias, particularly in progressive pernicious anæmia. They appear in specimens stained with eosin-methylene-blue mixtures, as purple, brownish, or bluish cells, their color, as a rule, being unevenly distributed, and in some instances only a part of the protoplasm exhibits this altered tinctorial reaction. The cytoplasm of nucleated red cells, especially of the megaloblast, often shows this change. Some authorities maintain that this abnormal staining quality is an indication of immature cell development, while others regard it as a sign of stroma degeneration. Oval or "ring-like" bodies reacting to basic dyes are occasionally observed in the red cells which some students assert are the remains of a nuclear structure. Red cells which contain granular areas having a basic stain affinity scattered through the cytoplasm, appearing in some corpuscles as a fine stippling and in others as coarse irregular granules, are described as cells showing *granular basophilia.* This condition is observed in severe anæmias, especially of the pernicious type, in leukæmia, and constantly in chronic lead poisoning. Some investigators regard it as an indication of degeneration of the cells, while others are inclined to view this feature as an evidence of nuclear fragmentation.

Nucleated Red Blood-cells. — Nucleated red blood-corpuscles are normally found in the blood during the early months of fetal life and in the blood-marrow of all individuals. Two principal types are found, normoblasts and megaloblasts.

Normoblasts. — This cellular element, a normal constituent of the bone-marrow of the healthy adult, is about the size of the normal erythrocyte, and consists of a single oval or round nucleus (rarely two or three), which reacts intensely to basic stains, while the cytoplasm has an acidophilic affinity like a normal erythrocyte. At times this cell contains an irregularly shaped nucleus or several may be noted in a single cell. The nucleus is often eccentrically placed, and sometimes extrudes from the cell or may be found free in the plasma. The occurrence of normoblasts in the circulation of the adult is generally regarded as a sign of rapid blood regeneration, well illustrated after a profuse traumatic hemorrhage, where

large numbers of these cells often are present in the blood, a condition which Von Noorden has termed "blood crisis." Cells having the diameter ranging from 4 to 6 microns, with a round or oval nucleus reacting sharply to basic dyes, and a shrunken irregular protoplasm, called microblasts, probably represent normoblasts having a degenerated cytoplasm. *Megaloblasts* vary in size between 11 and 20 microns in diameter, and consist of a large nucleus of loose texture staining feebly, surrounded by a comparatively small amount of cytoplasm. A clear hyaline space or ring sometimes separates the nucleus from the protoplasm, which not infrequently has a polychromatophilic reaction. Fetal bone-marrow normally contains megaloblasts. Most writers regard the presence of megaloblasts in the circulating blood of the adult an indication of a reversion of the marrow activity to an earlier type similar to that found in the fœtus. These cells are found in grave forms of anæmia, as typified in pernicious anæmia.

Blood-platelets.—Blood-platelets, or blood-plaques, are small, spherical, oval, or irregular bodies, having a pale yellowish color, and measure from 1 to 4 microns in diameter. They are not endowed with amœboid activity, and stain with both acid and basic dyes. Blood-plaques disappear rapidly after the blood is exposed to the air. Some writers consider these elements as being derived from fragmented red blood-cells.

Technic of Counting Blood-platelets (Method of Wright and Kinnicutt).—Two parts of cresyl blue (1 : 300) are mixed with three parts of potassium cyanide (1 : 14000) and rapidly filtered, then immediately used to dilute the blood 1 : 100. The blood must be from a free-flowing drop and the whole procedure must be carried out as quickly as possible. Variations under physiological and pathological influences are common. In many severe secondary anæmias, in leukæmia, in chlorosis, and in rheumatoid arthritis an increase is encountered, while in pernicious anæmia their number is generally reduced. In some of the specific infectious diseases, particularly in pneumonia and in bubonic plague, an increase is found, while in others, notably in erysipelas, in malaria, and in typhus fever, there is a decrease. The average per cubic millimetre at sea level is 302,000, and 340,000 at an altitude of 6000 feet—a percentage increase of 12.2. (Webb and Gilbert.) They are consistently increased in tuberculosis of man and guinea-pigs. A marked reduction is frequently seen in purpura and hæmophilia.

Hæmokonia.—In fresh unstained blood there are found, in the plasma, small, transparent, highly refractile bodies, not exceeding one micron in diameter, of spherical, oval, or dumb-bell shape, possessing active molecular motion, which are termed hæmokonia, or blood dust. These bodies are insoluble in ether or alcohol and do not stain with osmic acid. Their significance is as yet unknown; it has been suggested that they represent fragments of cells or free cell granules, as eosinophilic or neutrophilic granules.

Leucocytes.—The leucocytes, or white blood-corpuscles, in a wet preparation of fresh blood taken from a normal individual, appear as pale, colorless, nucleated cells, the greater number of which are granular and endowed with amœboid activity. Ehrlich's classification is generally adopted for clinical work. The following table includes the main varieties of leucocytes with their relative percentages present in the blood of the normal adult:

Polynuclear neutrophiles	60–70 per cent.
Eosinophiles	.5– 4 per cent.
Basophiles or mast-cells	.025–.5 per cent.
Small lymphocytes	20–30 per cent.
Large lymphocytes, hyaline cells, and transitional forms	4– 8 per cent.

In infancy the percentage of lymphocytes is greater than in adult life, while eosinophiles may reach as high as 14 per cent. in childhood.

Polynuclear Neutrophiles.—These cells, the diameter of which ranges between 7½ and 11 microns, have an irregular nucleus, appearing in various shapes, as in the form of the letters U, Z, S, and a finely granular protoplasm. The irregularly shaped nucleus, which is composed of enlargements or lobes connected by bands, reacts to basic dyes with marked affinity. The granules are fine, of an irregular outline, and absorb acid dyes (finely granular oxyphile cells). According to Ehrlich, the granules have a neutral staining property. The polynuclear neutrophiles possess amœboid and phagocytic properties.

Eosinophiles or polynuclear eosinophiles (coarsely granular oxyphiles) are about the size of or slightly larger than the normal erythrocyte, their diameter ranging from 7 to 10 microns. They possess a nucleus similar in structure and tinctorial qualities to that of the polynuclear neutrophile; their protoplasm contains coarse, highly refractile, oval or spherical granules, staining deeply with acid dyes. They are endowed with active amœboid qualities.

Basophiles or Mast-cells.—Under this term are classified leucocytes which have a lobed or twisted nucleus like that of the neutrophiles and a cytoplasm beset with very irregularly shaped basophilic granules of varying size. The granules are not colored with Ehrlich's triple stain, but may be plainly seen when treated with Leishman's or Ehrlich's dahlia mixture.

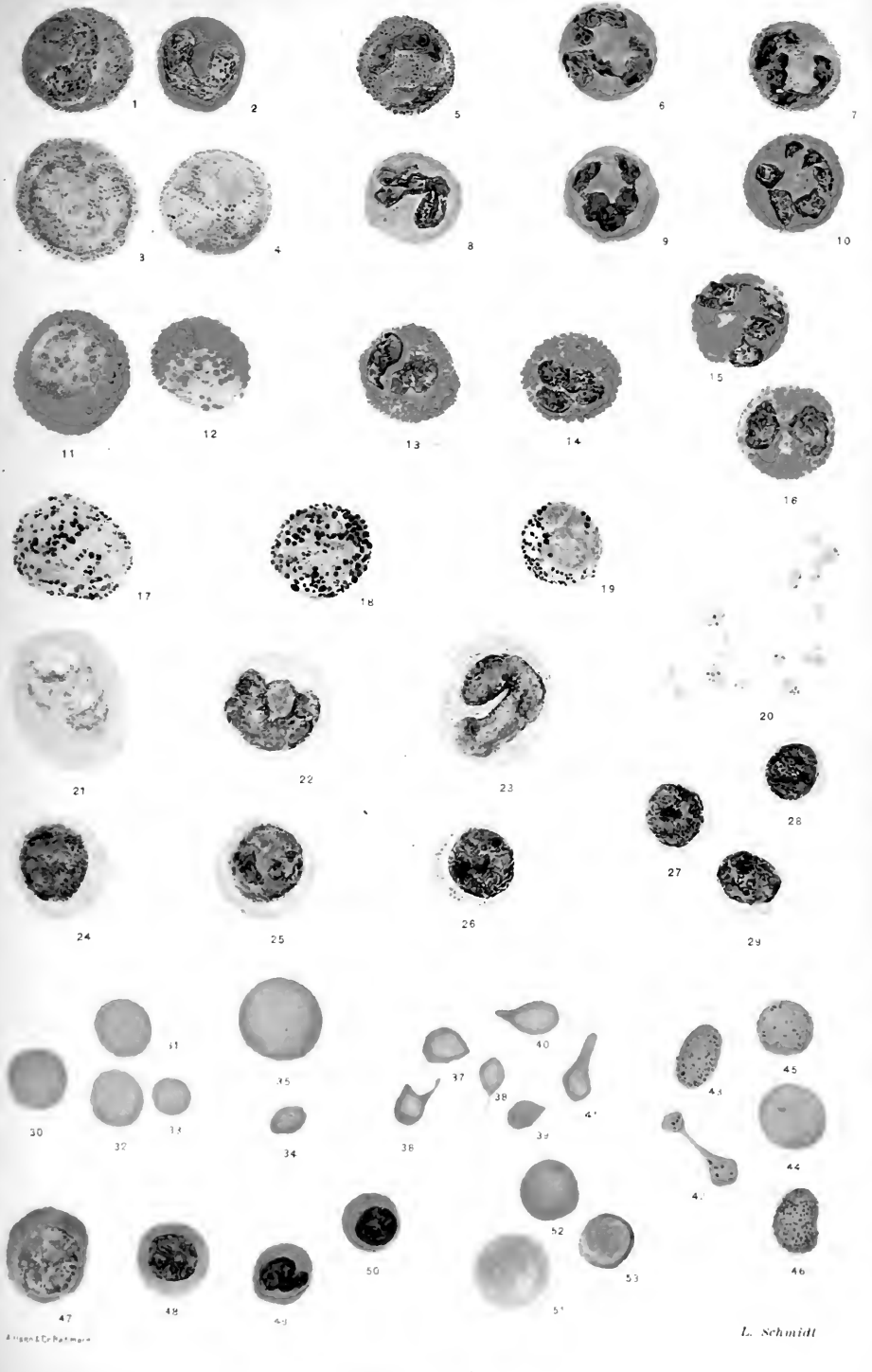
Small Lymphocytes. — These are essentially non-granular cells, the majority being about the size of the normal erythrocyte. They consist of a large circular or oval nucleus, which has a decided basic property and a relatively small amount of protoplasm, reacting feebly to basic and occasionally to acid stains. With Ehrlich's triple stain, the cytoplasm is colored a pale pink or gray, while with Leishman's eosin-methylene-blue mixture, a light blue, showing less basic affinity than the nucleus. Lymphocytes treated with Leishman's stain occasionally show a few fine pink granules in their cytoplasm. These cells are neither amœboid nor phagocytic.

Large Lymphocytes. — Several varieties of leucocytes are embraced under this heading—*lymphocytes proper of large size*, generally regarded as the product of lymphatic tissue, and *large mononuclear or hyaline cells*, probably of bone-marrow origin. A distinction cannot always be made between large lymphocytes and hyaline cells, since they resemble each other as to structure and tinctorial reactions. The nucleus of the latter cell is round or oval. The protoplasm of the lymphocyte has a slightly stronger basic affinity than that of the large mononuclear. From a clinical standpoint this differentiation does not appear important. The large lymphocytes have a relatively smaller nucleus than the small forms, and stain less intensely. *Transitional forms* closely resemble large lymphocytes and hyaline cells in size and staining qualities, but differ from

DESCRIPTION OF PLATE IV.

1. Neutrophile myelocyte.
2. Neutrophile myelocyte showing indentation of its nucleus.
3, 4. Neutrophile myelocytes.
5, 6, 7, 8, 9, 10. Polynuclear neutrophiles.
11, 12. Eosinophile myelocytes.
13, 14, 15, 16. Polynuclear eosinophiles.
17. Basophile myelocyte.
18, 19. Polynuclear basophiles.
20. Blood platelets.
21. Large mononuclear form.
22, 23. Transitional forms.
24, 25. Large lymphocytes.
26. Lymphocyte showing acidophilic granules in its protoplasm.
27, 28, 29. Small lymphocytes.
30, 31, 32. Normal erythrocytes.
33, 34. Microcytes.
35. Macrocyte.
36, 37, 38, 39, 40, 41. Poikilocytes.
42, 43, 44, 45. Erythrocytes containing basophilic granules.
46. Erythrocyte exhibiting polychromatophilia and granular basophilia.
47. Megaloblast.
48, 49, 50. Normoblasts.
51, 52, 53. Erythrocytes with polychromatophilic protoplasm.

(Leishman's Stain.)



L. Schmidt

(LEISHMAN'S STAIN)

these leucocytes in having an indented or distorted nucleus, resembling the form of the nucleus of some of the polynuclear neutrophiles.

Hyaline cells are said to possess active phagocytic and amœboid properties.

Myelocytes.—These cells, which are normal constituents of the bone-marrow and only present in the blood in pathological states, are in the main large cells supplied with a large circular, oval, or slightly indented nucleus, staining feebly with basic principles, and surrounded by a granular protoplasm.

These cells are classified into three groups, depending on the microchemical reaction of the cell-granules, namely into neutrophilic, eosinophilic, and basophilic varieties. The neutrophilic myelocyte is the commonest form. Neutrophilic granules of myelocytes do not stain as distinctly as those found in the polynuclear cells. Types intermediate between typical polynuclear neutrophiles and typical neutrophilic myelocytes are observed in some pathological conditions, especially myelogenous leukæmia and not infrequently in leucocytosis. Myelocytes often show indistinct granulations embedded in a feebly basic protoplasm; these forms are considered by some to be closely related to cells farther back in their ancestral development.

Atypical lymphocytes are not infrequently encountered in severe anæmias, particularly in lymphatic leukæmia. Some of these cells are almost devoid of protoplasm, while others contain a distorted nucleus. The term neutrophilic pseudolymphocyte has been suggested for those cells which have a round nucleus, rich in chromatin, surrounded by a narrow rim of protoplasm, filled with neutrophilic granules. These leucocytes may represent small myelocytes, as their color characteristics, excluding the strong basic nucleus, suggest neutrophile myelocytes. Türk employs the name "stimulation form" for moderately large leucocytes having a single round weakly basophilic nucleus, and a non-granular cytoplasm, which stains a brown color with the triple mixture.

Development of Leucocytes.—Most authorities regard the bone-marrow and the lymphatic tissues as the seats of leucocyte formation, the former being concerned in the development of polynuclear neutrophiles, eosinophiles, basophilic and hyaline cells, while the latter appear responsible for the production of lymphocytes. In the bone-marrow are found groups of cells, "leucoblastic areas," consisting of myelocytes surrounded by polynuclear elements, while erythroblastic zones are present principally along the margins of vascular spaces. It is conceded by most authorities that the polynuclear neutrophile is developed from the neutrophile myelocyte, the polynuclear eosinophile from the eosinophile myelocyte, and a basophilic cell from its parent marrow cell. In the transformation of the myelocyte into the polynuclear leucocyte, the nucleus undergoes condensation and lobulation, the size of the cell decreases, and the staining reaction of the nucleus and of the granules intensifies. The large mononuclear or hyaline and transitional leucocytes are probably formed in the marrow. The lymphocytes are derived from lymphatic tissue, the small form being the progeny of the large cell.

Number of Leucocytes. — Many circumstances affect the number of leucocytes in the circulation. The colorless corpuscles range between 6000 and 8000 per cubic millimetre in a healthy person; this standard is, however, subject to slight variations, beyond these limits, in certain physiological, and often to pronounced alterations in pathological states. The number is influenced by the condition of body nutrition, *e.g.*, in prolonged starvation low counts are conspicuous, by unequal distribution of the cells, by blood dilution and by blood inspissation. The most acceptable theory, advanced to explain the occurrence of an increase in the number of colorless elements (leucocytosis) in disease, points out that irritants acting in the tissues produce chemical substances which attract certain leucocytes towards the seat of mischief, and cause the leucocyte forming organs to pour out an increased number of cells. This attraction force has been termed "*positive chemotaxis,*" in contradistinction to a repelling action set up by some irritants called "*negative chemotaxis.*" The degree of leucocytosis depends mainly upon the intensity of the chemotactic force and the responding powers of the individual.

Leucocytosis.—The term leucocytosis or hyperleucocytosis designates an increase in the number of leucocytes in the peripheral blood over the normal standard. This increase, as a rule, involves a marked percentage gain in the polynuclear neutrophile elements with a fall in the percentage of other forms, but sometimes comprises a proportionate rise in all the varieties, or a percentage gain in the lymphocytes, eosinophiles, or basophiles. Leucocytosis is classified into special forms, depending on physiological or pathological disturbances; these types being further subdivided into special varieties, dependent upon certain etiological factors,—*e.g.*, digestive, inflammatory, toxic, malignant, and post-hemorrhagic leucocytosis. A rise in the number of polynuclear neutrophile cells is called polynuclear neutrophile leucocytosis; an augmentation in lymphocytes is termed lymphocytosis; an increase of eosinophiles is known as eosinophilia; the latter forms also being sub-grouped into (a) an absolute and (b) a relative increase; *e.g.*, absolute lymphocytosis is shown by a gain in the total count with a rise in the percentage of lymphocytes, while relative lymphocytosis implies a percentage gain without an increase in the total number of these cells. The terms absolute and relative as applied to leucocytosis are often misleading to the student.

Physiological Leucocytosis —The leucocytosis which depends upon physiological factors is, in the main, slight, of short duration, and commonly involves a proportionate increase in all of the forms of leucocytes, less often an absolute and a relative gain in the polynuclear neutrophile elements.

Leucocytosis of Digestion. — In nearly all healthy individuals, during the period of digestion, from one to four hours after taking food, a slight rise in the number of leucocytes is present, which generally consists of an absolute increase. Some claim that the gain principally involves the neutrophiles, while others assert the lymphocytes are responsible. In the new-born, leucocytosis of digestion is pronounced. In starvation and frequently in the morbid states associated with faulty nutrition, the number of white corpuscles decreases. The rapidity with which digestion leucocytosis manifests itself after taking food is regarded by some writers

as bearing a direct relation to the activity of the digestive function. In persons suffering from gastric ulcer, leucocytosis sometimes comes on very soon after taking food, pointing to rapid digestion; while in gastric cancer, it may be delayed or absent. This rule is not constant and little importance should be attached to digestion leucocytosis in diagnosis.

Leucocytosis Occurring during Pregnancy and after Parturition.—A moderate rise in the number of leucocytes occurs during the later months of pregnancy, and persists for about two weeks after parturition.

Leucocytosis of the New-born. — The high counts observed for about ten days after birth are attributed to blood inspissation and to the establishment of digestion leucocytosis. Higher counts are present in early childhood than in the advanced periods of life. Leucocytic oscillations of a very moderate character occur after exercise, massage, after cold or hot bathing, and after the use of electricity.

Pathological Leucocytosis. — Inflammatory and Infectious Leucocytosis.—The presence or the absence of a leucocytosis in many of the infectious and inflammatory diseases is a sign of considerable importance in diagnosis. Its clinical value is comparable in a measure with other signs, such as temperature range and pulse, certain physical signs, etc. This pathological increase, which is essentially a polynuclear neutrophile leucocytosis, is as a general rule encountered in acute local inflammations, as in furuncles, cellulitis, abscesses, in general sepsis, and in nearly all of the specific infectious diseases, except in uncomplicated cases of enteric fever, paratyphoid infections, tuberculosis, malaria, measles, German measles, influenza, leprosy, and Malta fever. High leucocytic ranges are often attributable to suppurative inflammation. Duration and height of fever have no direct relation to the leucocyte curve. Counts between 15,000 and 20,000 are common, while ranges above 30,000 are uncommon and above 50,000 very rare.

Preagonistic or Terminal Leucocytosis. — The exact nature of the leucocytosis which so often precedes death is still a mooted question; many investigators attribute this rise to terminal infections.

Malignant Leucocytosis.—Different opinions have been advanced to explain the gain of colorless elements so frequently noted in individuals suffering from carcinoma and sarcoma. Some writers contend that associated inflammatory disturbance about the growth or septic absorption from the tumor induces the leucocytic gain, while others hold that the direct effect of the tumor is the responsible factor. In rapidly spreading malignant growths, especially when metastasis has occurred, counts are generally high and much above those noted in slowly growing tumors; in sarcomata the gain is usually more pronounced than in carcinomata. The cellular rise in the majority involves mainly the neutrophiles, although lymphocytosis, especially in sarcomata, has been recorded. In cases of carcinoma of the stomach, leucocytosis is often absent.

Post-hemorrhagic Leucocytosis.—The gain in the white corpuscles which follows and persists for some days after a profuse hemorrhage is generally accounted for by an increased production and output of polynuclear neutrophiles. Some observers hold that the lymph (rich in cells) which passes into the blood after blood loss is the responsible factor.

A leucopenia, lasting for a few hours, precedes the leucocytosis due to hemorrhage. Leucocytosis is present in many cases of secondary anæmia.

Leucocytosis due to Toxic and Therapeutic Agents. — Among the substances capable of producing a rise in the leucocytic standard may be mentioned quinine, ether, chloroform, potassium chlorate, illuminating gas, salicylates, uric acid, and various organic extracts.

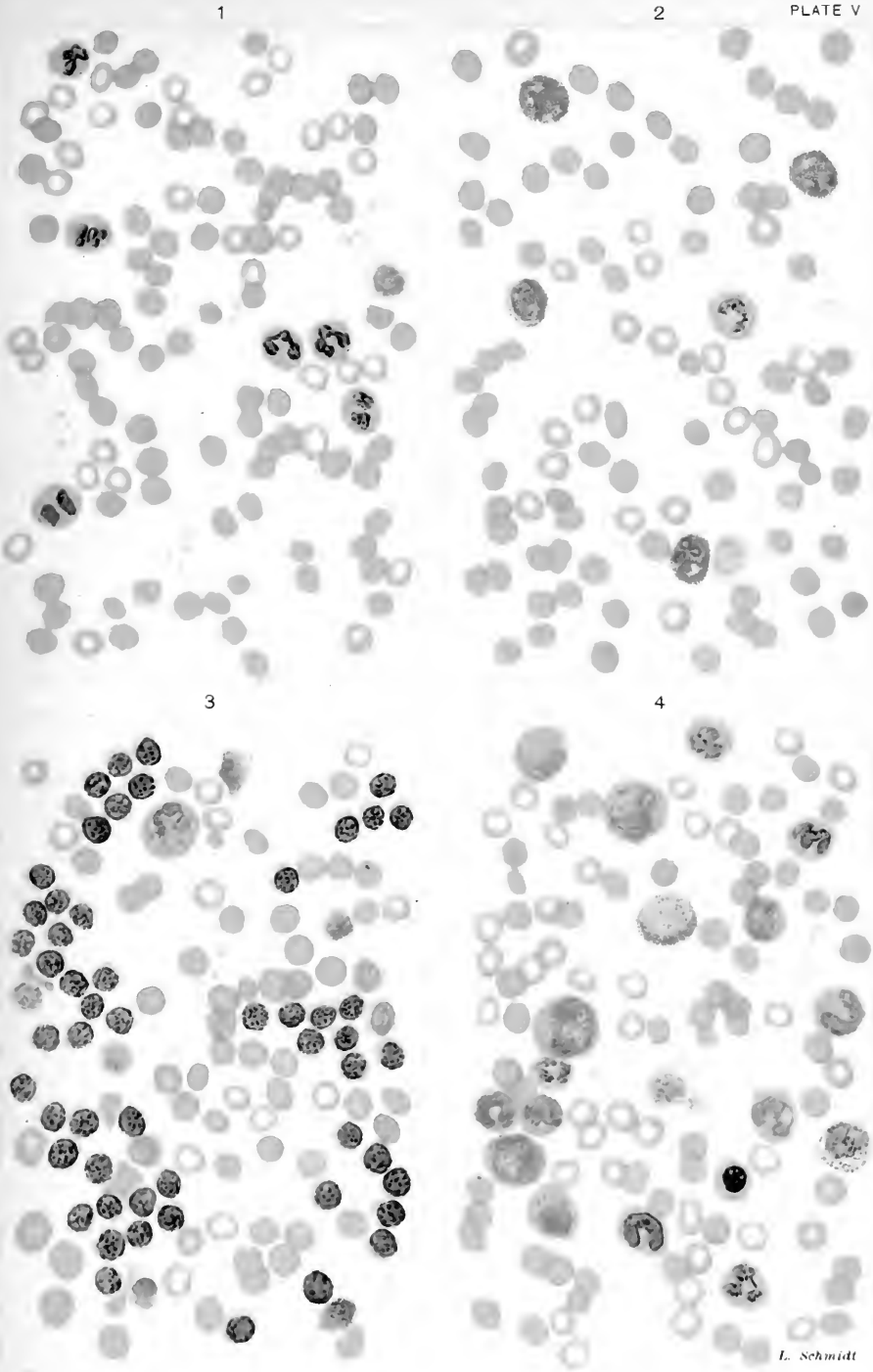
Lymphocytosis. — This condition, an increase of lymphocytes, is normal in infants and in young children. A relative lymphocytosis due to diminution of the polynuclear elements has been recorded in the following conditions: chlorosis, pernicious anæmia, severe secondary anæmia, and in some of the infectious diseases, as tuberculosis, enteric fever, malaria, and influenza. A lymphocytosis is sometimes observed in Hodgkin's disease, and often in children suffering from syphilis, anæmia, pseudoleukæmia infantum, rickets, whooping-cough, and gastro-intestinal diseases. Diseases of the lymphatic glands and spleen are sometimes attended by an increase in these cells. Lymphocytosis in most instances is a relative condition. Absolute lymphocytosis of high grade is a constant feature of lymphatic leukæmia. In myelogenous leukæmia the percentage of lymphocytes is reduced, although the total number is increased.

Eosinophilia.—An increase in the number of eosinophiles has been observed in the blood of normal infants, in anæmia pseudoleukæmia infantum, in splenomedullary leukæmia, in bronchial asthma, after hemorrhage, in diseases of the skin, as urticaria, lupus, eczema, leprosy, and pemphigus, after coitus, during convalescence from many of the acute infectious diseases, in bone diseases and in morbid states caused by intestinal worms, particularly in trichiniasis. Definite conclusions regarding the clinical significance of fluctuations in these cells in morbid states cannot be drawn from our present knowledge, although some diagnostic importance should be attached to the almost constant eosinophilia in trichiniasis, in ankylostomiasis, and in bilharzial infection.

Basophilia.—This term is used to express an increase in the number of basophiles in the circulating blood, which is frequently noted in splenomedullary leukæmia. Some writers have reported basophilia in splenic anæmia, in certain skin diseases, in acute bone inflammation, and in gonorrhœa. The clinical significance of basophilia remains unsettled.

Leucopenia.—A decrease in the number of leucocytes is termed leucopenia, or hypoleucocytosis, and may be brought about by physiological and pathological factors. A deficiency in the number of colorless cells is seen in starvation and in malnutrition, and almost constantly in the infectious diseases not associated with leucocytosis, as tuberculosis, malaria, measles, influenza, enteric fever, Malta fever, and German measles. Low leucocyte counts are common in pernicious anæmia, in chlorosis, in splenic anæmia, and in profound symptomatic anæmias.

Myelæmia.—The appearance of myelocytes in the blood, spoken of as myelæmia, points to rapid leucocyte proliferation in the bone-marrow. Myelocytes are often found in the circulation when leucocytosis is present. In splenomedullary leukæmia this condition is most striking, in pernicious anæmia, cholorsis, lymphatic leukæmia, Hodgkin's disease, and in profound secondary anæmias, a small number of myelocytes is not infrequent.



1. NEUTROPHILE LEUCOCYTOSIS.
2. EOSINOPHILIA.
3. LYMPHOCYTOSIS.
4. MYELÆMIA.

(LEISHMAN'S STAIN)

Parasites.—Bacteræmia.—Bacteria frequently gain access to the circulation, either from an infected area within the body or from without. Bacteria are seldom found by a microscopical examination of the fresh unstained blood; culturing methods being required to reveal their presence. From a clinical view-point the demonstration of bacteria in the circulation may be regarded as evidence of disease. The detection of micro-organisms in the blood is often essential in establishing a diagnosis of septicæmia, malignant endocarditis, puerperal sepsis, and pyæmia. In some of the specific infectious diseases the exciting principle has been isolated from the blood. In over 80 per cent. of the cases of enteric fever, Eberth's bacillus can be recovered from the blood by culture methods. The specific micro-organisms of paratyphoid fever, croupous pneumonia, anthrax, glanders, leprosy, influenza, plague, tuberculosis, and Malta fever have been isolated from the blood. In septicæmia or septicopyæmia staphylococci, streptococci, and gonococci have been separated from the blood.

Animal Parasites. — From our present knowledge it is impossible to fix a sharp dividing line separating some of the lower vegetable and animal parasites. Investigators seem to favor placing the Treponema pallidum and the spirochæta of relapsing fever among the animal parasites. For a description of the animal parasites found in the blood, the reader is referred to the section which deals with diseases caused by animal parasites.

Method of Examination for Malarial Parasites.—Fresh, unstained blood is more suitable for study than stained blood, because it enables the examiner to observe the activity of the parasites. The most desirable time for conducting the examination is during the period when pigmented forms are present. The intracellular pigmented bodies are usually most abundant about 6 or 8 hours before a paroxysm. Considerable experience is necessary before the various types can be differentiated; especially is this true of hyaline unpigmented forms. The skilled microscopist must often make a prolonged search before parasites are detected in malarial blood. The fresh blood is prepared in the usual manner, between a coverglass and slide, and the examination is made with an oil-immersion lens with moderate illumination. When the examination of fresh blood is impracticable, dried specimens treated by one of the different forms of the Romanowsky stain as variously modified by Leishman and others (Wright, Hastings) constitute a satisfactory means of direct diagnosis. The effectual administration of quinine reduces the number of parasites and causes their disappearance from the peripheral circulation. In a suspected case the examination of the blood should precede the institution of treatment.

VI.

THE EXAMINATION OF THE URINE.

The essential diagnostic principles of urinalysis, coupled with certain methods of examination required for general clinical work, are detailed in the following section. Tests suitable for the general practitioner must in the main be simple and easily applied. The importance of the findings obtained by careful and systematic studies of the urine, when correctly interpreted and given their proper place in a symptom-complex, can scarcely be overestimated in diagnosis. In a considerable group of morbid states the urinary picture is absolutely necessary in establishing a final diagnosis, while in a large number of cases the results of the examination form a link in the chain of symptoms completing the diagnosis. Negative reports in some instances are essential for the solution of certain clinical problems. For an exhaustive account of the urine, which does not fall within the scope of this book, the reader is referred to special treatises on this subject.

PHYSICAL EXAMINATION.

Amount.—The daily standard for a healthy adult, as estimated by different authorities, varies between 900 and 2000 c.c. Most observers fix the amount between 1000 and 1500 c.c., although temporary oscillations beyond these limits, of slight, moderate, or even of an excessive degree, are often physiological. The urinary secretion is greater during the day than through the night, more abundant in cold than in hot weather, and relatively more active in children and infants than in adults. Polyuria depends in the main on, (a) increased ingestion of water, (b) heightened blood-pressure, and (c) on increased activity of the renal epithelium, while oliguria results from (a) lessened consumption of water, (b) lowered blood-pressure, and (c) impaired function of kidney epithelium.

Specific Gravity.—In health the specific gravity ranges between 1.015 and 1.025 while the daily output of urine is within normal limits. In general terms the specific gravity is a fair index of the bulk of solids eliminated. An approximate estimate of the weight of urinary solids expressed in grammes for a thousand cubic centimetres of urine may be determined by multiplying the second and third decimal figures of the specific gravity by two. Pathologically, wide variations, as low as 1.002 and as high as 1.040, are frequent. Ranges above 1.050 are extremely uncommon. The volume of urine and the specific gravity in the main fluctuate in an inverse manner; therefore, without a knowledge of the daily output the density has practically no clinical significance. A correct specific gravity determination can only be made from a mixed twenty-four hours' sample. In diseases associated with polyuria lowered ranges are the rule. A notable exception is found in the case of diabetes mellitus, in which the increased density is caused by the presence of glucose. A diminished volume of urine of low specific gravity is seen in a number of chronic diseases, and

often in cases of œdema. Large urinometers are preferable to small instruments for measuring the specific gravity, since the divisions of the scale can be read with greater accuracy. The large amount of urine necessary to float these hydrometers is, however, an objectionable feature. This may be overcome by estimating the density with a set of two or three pycnometers of moderate size, each of which represents a portion of the scale. In measuring the specific gravity the following precautions are essential for accurate results: The hydrometer should be placed into cool urine, the surface of which is free from foam; the instrument should not touch the sides of the hydrometer jar.

Color.—Normal urine owes its color to urochrome and urobilin, chiefly to the former, while the tints of many pathological urines depend upon such substances as biliary pigment, hæmoglobin, methæmoglobin, hæmatin, hæmatoporphyrin, melanin, indican and alkaptone. After the ingestion of iodine, phenol, salol, senna, santonin, and methylene blue, the urine often has an abnormal hue. The shade of the color depends chiefly upon the amount of urinary water, so that concentrated specimens are usually dark, while those of low specific gravity are generally pale. An exception to this rule is noted in diabetic urine, which is light yellow or pale greenish-yellow, of increased density and of excessive quantity. In acute febrile diseases and pernicious anæmia the urine is high colored, while in chlorosis, diabetes insipidus, and contracted kidney it is pale. Bile pigment is responsible for dark yellow, yellowish-green, dark brown, and rarely, black urine, which on shaking develops a yellowish foam; blood pigment stains the urine bright red, dark red, reddish-brown, or rarely, black. Melanotic pigment imparts a brown or black appearance which generally develops some time after the urine is voided, but occasionally is noted in the fresh specimen. A similar color occurs in alkaptonuria, which condition can be differentiated from melanuria by testing with Fehling's solution; the alkaptone bodies reduce the copper salt, while the latter gives a negative reaction. The ingestion of phenol and its allied compounds may cause a greenish-black discoloration, of methylene blue a green or blue urine, of santonin a yellow, and of rhubarb an orange-colored urine. A milky appearance is noted in chyluria and at times in phosphaturia and pyuria. The presence of hæmatoporphyrin may impart a dark red color.

Transparency. — Normal urine immediately after being voided is generally clear; on cooling it occasionally becomes turbid, due to precipitation of urates or phosphates. Turbidity, associated with a sediment, is one of the characteristic features of many abnormal urines, and may depend upon an excess of urates or phosphates, or on the presence of epithelial elements, pus-cells, red blood-corpuscles, chyle, or bacteria.

Odor.—This property of the urine, although of little clinical significance, except in a few instances, occasionally attracts the attention of the patient who suspects that an abnormal state of the kidneys exists. The odor of normal urine is sufficiently familiar to require no special description. Urine decomposed by bacterial growth within the bladder, or after it has been voided, emits an ammoniacal stench. Acetone, when present in large amount, may give the urine a sweetish, fruit-like odor. The ingestion of turpentine, terebene, asparagus, and onions imparts peculiar odors.

Reaction.—A normal, mixed twenty-four hour sample in nearly every instance is acid, while individual specimens passed during the day vary considerably in reaction. For clinical purposes the reaction may be determined with litmus paper. An alkaline reaction after a heavy meal is attributed to the increased alkalinity of the blood during gastric digestion. The ingestion of food rich in vegetables, or the administration of tartaric, citric, or acetic acid lessens acidity, or produces alkalinity, while a diet rich in meat or the taking of mineral acids intensifies the acidity. The reaction of normal urine is held to be due to diacid phosphate; Folin, however, contends that free organic acids are in part responsible. The total acidity of a twenty-four hour collection of healthy urine is equal to from 1.5 to 2.3 grammes of hydrochloric acid. Alkalinity is caused by the presence of alkalies in excess of acids. An amphoteric reaction depends on a balance existing between the acid and basic equivalent of the urinary salts. Urine that has been exposed to the air for some time becomes alkaline from ammoniacal decomposition. Alkaline urine is frequently seen in cystitis, but in a number of cases of bladder inflammation, both acute and chronic, the urine is acid. A strongly acid urine occurs in gout, diabetes, rheumatic fever, in some varieties of nephrolithiasis, in leukæmia, in intestinal and stomach diseases associated with diminished or abolished gastric secretion, in scurvy, in chronic nephritis, and often in febrile states. Lowered acidity, and occasionally alkalinity, is seen in anæmia, notably pernicious anæmia and chlorosis, following the crisis of pneumonia, after blood transfusion with saline solution, in hæmaturia, and when transudates are rapidly absorbed.

Sediments. — Occasionally deposits of uric acid, amorphous urates, carbonates and phosphates, and invariably those consisting of pus, epithelial and red blood-cells, are significant of morbid states. The presence of a sediment of uric acid crystals, amorphous urates, or phosphates need not, and as a rule does not, imply an increased output of these salts, but may depend on changes in the reaction of the urine.

MICROSCOPICAL EXAMINATION.

Microscopical examination of sediments is of cardinal importance in urinalysis. A sediment best suited for this method of study should be secured from a fresh specimen of urine by centrifugalization. When the examination cannot be made immediately after the urine is voided, it is advisable to add an antiseptic to it, such as a little powdered camphor, a few drops of formaldehyde solution, or thymol, in order to prevent decomposition.

Crystalline and Amorphous Substances Present in Acid Urine.—Calcium Oxalate.—Crystals of calcium oxalate are found in many sediments and appear in various sizes, mostly in the form of colorless octahedra, generally designated "envelope crystals," sometimes as dumb-bell figures and rarely as oval disks. These crystals are soluble in hydrochloric acid, but not in acetic acid or sodium hydrate. Normal urine may contain these crystals, after the eating of tomatoes, asparagus, garlic, rhubarb, or oranges. After the ingestion of bicarbonate of soda, in certain forms of gastro-intestinal neuroses, in jaundice, in phthisis, and in diabetes mellitus, oxa-

late crystals are sometimes noted. Renal calculi composed of this substance are not uncommon. Oxaluria can only be regarded as pathological when a chemical examination shows an increased quantity of oxalic acid, provided the factors responsible for its occurrence in health can be excluded.

Uric Acid.—This substance, as a rule, crystallizes in the form of whetstone shaped crystals, arranged singly or in clusters, and occasionally in the form of dumb-bell figures, or as rhombic plates. These crystals vary considerably in size, and their color ranges from a pale yellow to a dark brown. Uric acid crystals dissolve in a sodium hydrate solution, and when this test is followed by the addition of hydrochloric acid to the alkaline solution, rhombic plates appear. Uric acid crystals are frequently seen in the urine when the uric acid output is normal or even decreased, since this substance is more readily soluble in warm than in cold solution. Urinary inspissation is another factor which determines precipitation. In leukæmia,

Fig. 107.—Calcium oxalate crystals.

Fig. 108.—Uric acid crystals.

and during or immediately following acute gout, the urine contains an excess of uric acid, and may reveal an abundant precipitate of these crystals (brick-dust sediment). Calculi formed of uric acid are among the most common renal concretions. When amorphous granules of sodium and potassium urate occur in the urine in abundance, they impart a turbidity to it, which is often associated with a light yellow or pink sediment. This precipitate disappears upon heating the urine to a temperature of 50° C.; on the addition of hydrochloric acid to the urine, amorphous urates are converted into uric acid crystals. Amorphous sediments of urates are frequently present in scanty, concentrated urines, such as occur in fevers.

Calcium sulphate is seen in the form of long, transparent colorless needles, or elongated platelets, arranged singly or in crystalline masses. They are insoluble in ammonia, acetic acid, and alcohol. Von Jaksch found these crystals in association with triple phosphates and calcium carbonate in the urine of a patient who showed a tendency to calculus formation. No special clinical significance has as yet been attached to the presence of these crystals.

Hippuric acid occurs as rhombic prisms or slender needles arranged separately or in clusters. These crystals are soluble in ammonia and insoluble in hydrochloric acid. They have been noted, though very infrequently, in febrile diseases, in diabetes, and after the ingestion of benzoic acid, salicylic acid, cranberries, mulberries, blueberries, and prunes.

Bilirubin is found as fine needles arranged in clusters or rhombic plates having a yellow or ruby color, or as an amorphous substance. This sediment is soluble in sodium hydrate and chloroform; on treating the crystals with nitric acid a green color appears about them. Von Jaksch contends that the presence of crystals of bilirubin in the urine, as a rule, points to antecedent hemorrhage into the urinary tract or to the rupture of an abscess. Their presence, either free or imbedded in cells or tube-casts, has been recorded in acute nephritis, chronic interstitial nephritis, amyloid kidney, jaundice, acute yellow atrophy of the liver, hepatic cirrhosis, phosphorous poisoning, carcinoma of the bladder, and after the rupture of a suppurating hydatid cyst into the urinary tract.

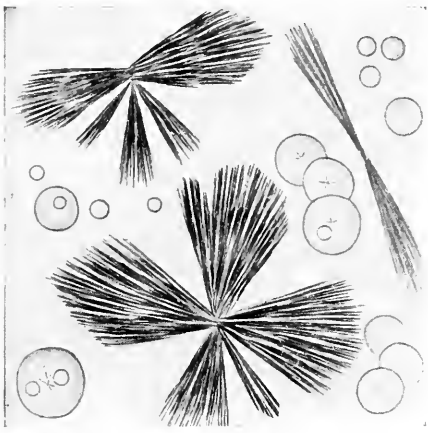


Fig. 109.—Leucin spheres and tyrosin crystals.

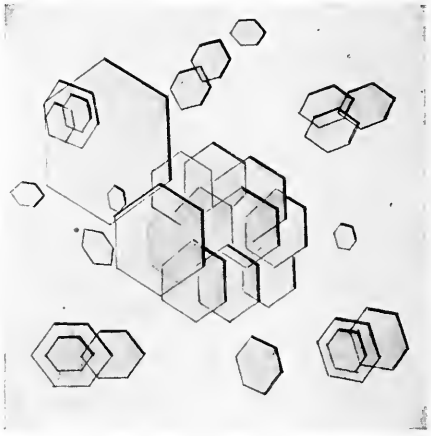


Fig. 110.—Cystin crystals.

Leucin and Tyrosin.—These substances are never found in normal urine. They are generally held in solution unless present in considerable quantities, when they separate in a crystalline form. Their precipitation can be brought about by treating the urine with an excess of basic plumbic acetate; then filtering; and to filtrate hydrogen sulphide is added to remove the excess of lead acetate. The filtrate is then evaporated to a small volume. Absolute alcohol is used to remove traces of urea. The insoluble residue is finally extracted with alcohol containing a little ammonia. Leucin and tyrosin will precipitate in this concentrated solution. Leucin presents the appearance of spheres of varying sizes, sometimes termed "leucin balls." These spheres have a brown color and show delicate lines radiating from their centre to the periphery. Leucin spheres are insoluble in ether. Tyrosin crystals are noted in the form of slender needles, frequently grouped in bundles. They dissolve in ammonia and hydrochloric acid, but are insoluble in acetic acid. Leucin and tyrosin occurring mostly

together have been observed almost constantly in acute yellow atrophy of the liver, phosphorous poisoning, and Weil's disease, occasionally in catarrhal jaundice, cholelithiasis, cirrhosis and cancer of the liver, enteric fever, gout, and diabetes, and rarely in a limited number of other conditions.

Xanthin is found in the form of colorless crystals resembling those of uric acid in outline. They are soluble in ammonia. These crystals are rare ingredients of urinary sediments; calculi consisting of xanthin have been found by some investigators.

Cystin crystals are six-sided colorless plates, which are soluble in ammonia and insoluble in acetic acid and water. Von Jaksch recommends the following microchemical test for their detection: A drop of hydrochloric acid is added to the urinary sediment. When the acid comes in contact with cystin, there develop prismatic crystals (hydrochlorate of cystin)

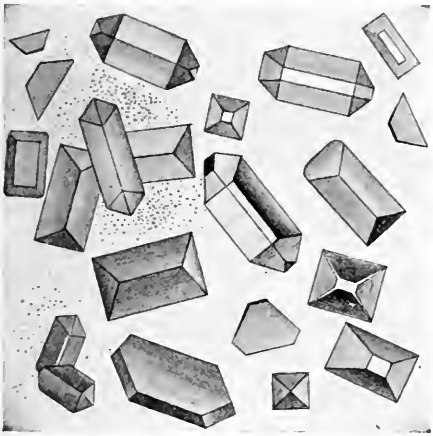


Fig. 111.—Triple phosphate crystals

Fig. 112.—Neutral calcium phosphate crystals.

which are grouped in masses suggesting a rosette form. Cystinuria is a rare condition. In some cases it is unattended with symptoms, while in others it is responsible for calculus formation.

Soaps of lime and magnesia consist of needles arranged in bundles or sheaves, sometimes radiating from a central point forming a sphere. They resemble tyrosin crystals in form and arrangement. These crystals are rarely found. They have been noted in septicæmia.

Fat globules can be recognized by their highly refractive appearance and solubility in ether. Among the conditions in which lipuria is seen, may be mentioned, acute and chronic parenchymatous nephritis, diabetes mellitus, bone disease and injury, chyluria, phosphorus poisoning, and certain diseases of the liver and pancreas.

Crystalline and Amorphous Substances found in Alkaline Urine.—Ammoniomagnesium phosphate or triple phosphate crystals are colorless and vary considerably in size and appearance. In their most characteristic form they occur as the so-called "coffin-lid" crystals; others resemble fern leaves in outline, while some are arranged in the shape of

the letter "X." Triple phosphate crystals are found in association with amorphous deposits of phosphates, carbonates, and at times with ammonium urate.

Neutral calcium phosphate occurs in alkaline, faintly acid, or amphoteric urine in the form of sheets or needle-like crystals, the latter being arranged singly or in masses forming dumb-bell or star-shaped figures. These bodies are soluble in acetic acid.

Neutral magnesium phosphate crystals are colorless, refractile, elongated plates with irregular or bevelled edges; they are soluble in acetic acid.

Calcium carbonate crystals are found in alkaline urine associated with amorphous carbonates. They are dumb-bell shaped bodies which dissolve in acetic acid with the liberation of gas.

Amorphous phosphates of calcium and magnesium and amorphous carbonates are of common occurrence in alkaline urine. The addition

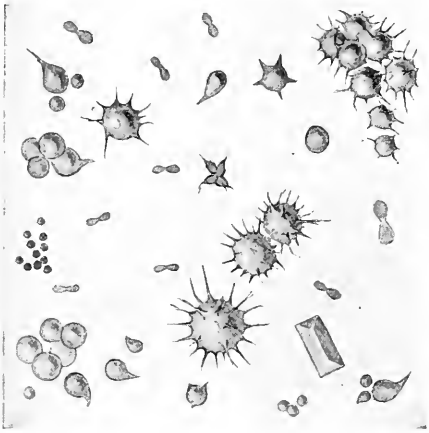


Fig. 113.—Ammonium urate crystals.

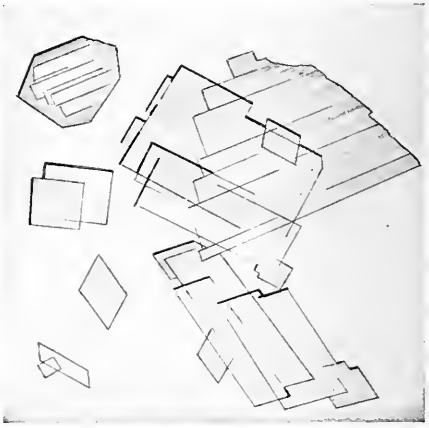


Fig. 114.—Cholesterin crystals.

of a fixed alkali to urine will precipitate amorphous phosphates and carbonates. On heating urine having a low acid or alkaline reaction, a white cloud similar to that produced by albumin appears which consists of phosphates or carbonates. On the addition of acetic acid, phosphates and carbonates are dissolved. The solution of the latter is attended with the evolution of gas. Amorphous phosphates or carbonates are often responsible for a turbid urine with a heavy sediment. Microscopically, these bodies appear as colorless, coarse granules, which are soluble in acetic acid. Phosphatic sediments are occasionally symptomatic of certain types of dyspepsia, of neurasthenia, of diseases associated with marked gastric acidity, and of some cases of cystitis. A deposit of phosphates does not of itself indicate an increased output of phosphoric acid; this can only be determined by quantitative analysis.

Ammonium biurate appears as dark brown spheroidal bodies from the surface of which spicules project, the so-called "thorn-apple" or "hedgehog" crystals, and in the form of coarse yellow needles grouped in clusters. Ammonia biurate may be associated with triple and amorphous phosphates.

Acetic acid will cause solution of these crystals with the formation of uric acid. Ammoniacal fermentation of the urine, occurring in the bladder or after it has been voided, is responsible for the precipitation of ammonium biurate.

Cholesterin crystals occur as colorless thin plates. They are rare constituents of urinary sediment and have been observed in hydatid cystic kidney, pyonephrosis, hydronephrosis, and cystitis.

Indigo, a rare ingredient of the urinary sediments, is found in the form of a blue crystalline body consisting of needles grouped in a stellate manner or as rhombic plates, and also as bluish amorphous granules. The amorphous material is not infrequently present in decomposing urine. Indigo is a rare constituent of urinary calculi.

Cellular Deposits. — Epithelial Cells. — Epithelial cells in small numbers, not sufficient to impart a sediment or cloudiness to the urine, can be found in every specimen. In many instances their number is so large as to justify a diagnosis of an inflammatory, atrophic, or degenerative lesion involving the genito-urinary tract. The predominance of one or several of the types of epithelial elements, unless correlated with other clinical data, has little significance. It is impossible to locate definitely a lesion of the genito-urinary tract from the morphological characters of epithelium alone. This difficulty is apparent when we consider, (1) similarity of many of the cells of different parts of the tract, notably those derived from the pelvis of the kidney, ureters, and bladder, (2) alterations in the shape of these delicate formations due to the action of the urine, and (3) the influence exerted on these cells by morbific factors, such as necrosis, pressure, etc. Therefore, little importance should be attached to their histological structure. Round cells, having a relatively large nucleus, are derived from the tubular structures of the kidney and the deeper layers of the renal pelvis. When casts are beset with round cells it points to a renal origin of this epithelium. An abundance of round cells in the absence of casts, especially when pus-cells and subjective symptoms pointing to pyelitis are present, is suggestive of origin from the renal pelvis. This opinion is strengthened by the presence of polygonal and conical cells, some of which have a tail-like elongation of their protoplasm; these cells are often arranged in a stratified manner. Round cells are also derived from the male urethra, while small conical and polygonal cells originate in the superficial layers of the pelvis of the kidney. Cylindrical cells with

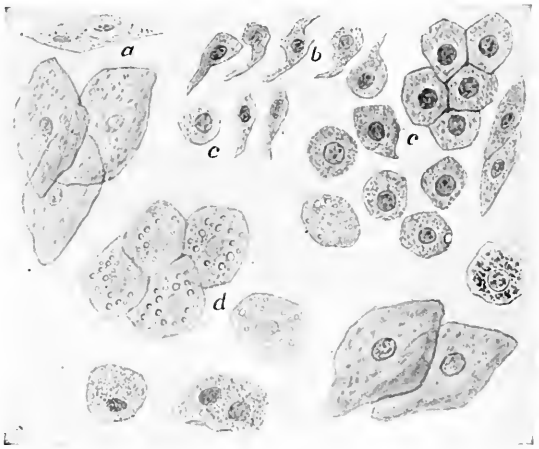


Fig. 115.—Epithelial cells. *a*, flattened cells; *b*, conical cells with tail-like prolongations; *c*, round and polygonal cells; *d*, degenerated cells.

bluntly pointed ends are found in the superficial layers of the male urethra. Flattened, oval or circular, or polygonal cells line the superficial layers of the ureter, bladder, prepuce, fossa navicularis, and vagina. Large squamous cells are generally derived from the vagina or prepuce. Protoplasmic degeneration of the epithelial cells is extremely common. A final diagnosis should never depend on the characters of the epithelia in the absence of clinical findings.

Leucocytes.—A careful search in every normal or morbid specimen of urine will reveal a few leucocytes. The action of the urine upon these cells causes alterations in their structure. In acid urine they have a distinct nucleus, while in alkaline urine their protoplasm is swollen and cloudy, obscuring the nucleus. By treating a specimen of urine having a weakly acid or alkaline reaction with acetic acid, the nuclear outline becomes sharply marked. The leucocytes stain a mahogany brown with a solution of iodo-potassic iodide (glycogen reaction), while epithelial cells are tinted a light yellow with this reagent. Pus-cells in considerable or large numbers frequently indicate inflammatory disease of some part of the genito-urinary tract. They occur in renal hyperæmia, nephritis, abscess and tuberculosis of the kidney, pyelitis, urethritis, cystitis, prostatitis, epididymitis, and orchitis. A leucorrhœal discharge is a common cause of pyuria. Urine containing many pus-corpuscles generally gives a positive reaction for albumin. Renal and extrarenal albuminuria may coexist.

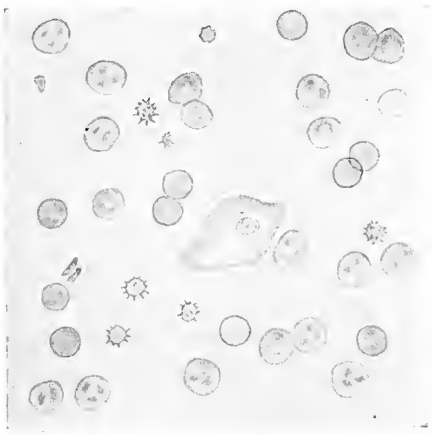


Fig. 116.—Red blood-cells and leucocytes.

Red Blood-cells.—Hæmaturia, or the presence of red corpuscles in the urine, is always pathological except during menstruation. In certain uterine diseases attended with bloody discharge some of the erythrocytes may be washed into the urine. A microscopic inspection serves to recognize red blood-cells in almost every instance, so that chemical tests for their detection are rarely required. In some urines the red cells are unaltered, while in others changes in their structure are found, such as decided shrinkage of the cell, or crenation, or they may be partially or completely decolorized, and appear as pale yellow disks or as faintly outlined rings (phantom corpuscles). The quantity of blood may be sufficient to tinge the urine pale or dark red, but in many cases the amount is so small that the microscopic test is essential in the diagnosis of hæmaturia. When the erythrocytes are intimately mixed with the urine, this suggests a hemorrhage in the kidneys, renal pelvis, or ureters. The presence of dehæmoglobinized corpuscles is noted in kidney lesions, such as congestion and inflammation. Unaltered blood or blood-tinged urine passed at the beginning of micturition is of urethral origin; on the other hand, when blood appears at the end of urination, its source is generally the neck of

the bladder. Bleeding may cause coagula of certain shapes; cylindrical clots of large size suggest urethral hemorrhage, those of small diameter may indicate ureteral hemorrhage, while irregular clots often form in the bladder. Neither the morphological characters of red blood-cells nor the size and outline of clot can be relied upon to definitely determine the site of a hemorrhage, unless these findings are supported by other clinical data. The use of the cystoscope, urethral and ureteral catherization, and examination with X-rays, especially for renal calculus, afford valuable adjuncts in the diagnosis of hæmaturia. An albumin reaction is invariably obtained when red blood-cells are abundant in the urine, but when only small numbers exist, a negative test is the rule. The quantity of albumin is proportionate to the amount of blood.

Tube-casts.—These are cylindrical bodies moulded in the uriniferous tubules. Their structure is variable, and may consist of a hyaline or waxy material, of cellular bodies, of granular elements, of fat globules, and in rare instances of bacteria or of amorphous substances.

Hyaline Casts.—These, by far the most common, are slightly refractile, transparent, of regular outline, with rounded ends. They are invisible in a brightly illuminated field on the microscope, so that it is necessary to cut off much of the reflected light with the iris diaphragm in order to bring out their outline. Epithelial cells, leucocytes, red blood-corpuscles, and granules frequently beset these casts, and, indeed, it is not uncommon to find adherent cells or granules so numerous that the hyaline material is obscured. Casts covered with granules are termed hyalogranular. This appearance may make it impossible to distinguish hyaline casts from those composed principally of granules. Clinically, however, the significance of hyaline casts coated with granules and those composed wholly or mostly of granules is identical. It should be remembered that a sharp distinction cannot be drawn between these forms. Hyaline casts are soluble in acetic acid.

Waxy casts appear as highly refractile, sharply defined, colorless or yellowish cylinders, showing a tendency to transverse fragmentation. Like hyaline casts they may be studded with cells or granules. They may exhibit an amyloid reaction, but this is no criterion that lardaceous renal disease exists, but, on the contrary, amyloid disease of the kidney is, as a rule, not associated with casts giving this reaction. Not infrequently, casts are observed that cannot be definitely classified as belonging to the waxy or hyaline varieties.

Granular casts are composed of fine or coarse granules. Cells forming leucocytic or epithelial casts may show a decided granular protoplasm, so that it becomes difficult to distinguish their outline; these types constitute border line varieties between cellular and granular casts. Clinically, this is of little moment, since the granular or fatty casts represent products of degenerated cells. Acetic acid dissolves granular casts.

Fatty casts consist of fat globules derived from degenerated cells. Ether dissolves fatty casts.

Epithelial casts are made up of renal epithelial cells, many of which present degenerative changes. These casts may have tubular form.

Leucocytic casts consist of white blood-cells. They are generally recognized at a glance, but should uncertainty arise as to the character of

the cells forming these cylinders, this doubt can be settled by treating the specimen with a droplet of acetic acid, which clarifies the protoplasm of the leucocytes and causes the nucleus to become distinct.

Fig. 117.—Tube-casts 1, 2, 3, 5, hyaline casts; 4, 6, hyaline casts beset with epithelial cells; 7, hyaline cast—one end of which is coated with fine granules; 8, hyaline cast beset with leucocytes; 9, finely granular cast; 10, coarsely granular cast; 11, 12, 13, waxy casts; 14, fatty cast; 15, 16, epithelial casts; 17, blood-cast; 18, leucocytic cast.

Blood-casts consist of erythrocytes, many of which may be altered by crenation or dehæmoglobinization. Pus- and blood-casts are rarely encountered. Casts formed of hæmoglobin, of bacteria, or of urates are rare.

Cylindroids are of two forms; one variety appears as long twisted or curved ribbon-like structures, composed of mucus, and therefore insoluble in acetic acid. This cylindroid is readily distinguished from true hya-

line casts, because of its length and flattened appearance. A second group consists of elongated cylindrical bodies. They show considerable variation in their short diameter, and are composed of a hyaline material. This cylindroid often tapers into a long thread-like tail. Some of the latter variety closely resemble hyaline casts, but can generally be distinguished from glassy casts by their irregular diameter. The material composing the latter type is soluble in acetic acid. Some authorities contend that this form of cylindroid has the same clinical significance as the hyaline cast. The mucous cylindroids are in the main formed in the bladder.

Clinical Significance of Tube-casts.—Tube-casts, especially of the hyaline variety, are often found in the urine of morbid states and occasionally of apparently healthy persons. Some observers maintain that their presence in the urine of so-called healthy persons can be explained by temporary circulatory disturbances, such as result from violent physical exercise or from overstimulation, as with alcohol. These circulatory derangements, although of a temporary nature, cannot be regarded as strictly physiological; therefore, the presence of casts under such circumstances reflects an abnormality of the renal function. The finding of casts over a long period generally warrants a diagnosis of structural changes in the kidneys. The number of casts present in a specimen of urine is sometimes an index of the extent or severity of renal involvement. In acute diffuse nephritis their number is generally large, in chronic parenchymatous nephritis they are usually fairly abundant, while in the interstitial form only a small number is noted. In passive renal congestion, amyloid disease, and in the degenerations attending febrile diseases, casts are generally few in number, but occasionally plentiful. The size of casts varies considerably. Large cylinders at times exceed 1 mm. in length. The size of tube-casts has no special diagnostic significance. The predominance of one or several varieties of casts may be of value in deciding the character of a renal lesion. Hyaline casts do not signify any special morbid change, as they occur under a variety of circumstances. Often

FIG. 118.—Cylindroids. 1, Cylindroids resembling hyaline tube-casts; 2, cylindroids stippled with granules; 3, ribbon-like mucous cylindroids; 4, spiral form of cylindroid.—Modified from Emerson.

they appear when there is only a slight functional derangement of the kidneys, but they are invariably present in organic renal disease. Waxy, granular, epithelial, and fatty casts point to degeneration of the renal parenchyma, while pus-casts may indicate purulent kidney disease. Blood-casts signify hemorrhage.

Spermatozoa and Testicular Casts.—Spermatozoa are found in the urine after coitus pollution, and rarely after convulsions. The urine which contains spermatozoa occasionally reveals testicular casts. These casts closely resemble renal casts. They can, however, be distinguished from the latter, since they occur only in the first part of the urine voided, while renal casts are present in the entire specimen. Their recognition depends mainly upon the finding of spermatozoa with these casts in the first urine of a two-glass test, and an absence of both of these elements in the second specimen of urine.

Irregular shreds and ribbon-like threads (Tripperfäden) are seen without magnification in the urine after acute gonorrhœa and in chronic urethritis. They consist of shreds of coagulated mucus, to which are generally adherent leucocytes and epithelial cells.

Bacteria; Animal Parasites.—Bacteria.—The presence of bacteria in abnormal urine depends upon (1) contamination of urine after it is voided, (2) existence of infectious lesions of the genito-urinary tract or communicating with it, and (3) elimination of bacteria from the blood by the kidneys. In large numbers bacteria impart turbidity to the urine, which does not clear up completely by centrifugating or by passing the urine through filter-paper.

The Micrococcus ureæ is considered responsible for ammoniacal fermentation. The colon bacillus, tubercle bacillus, typhoid and paratyphoid bacillus, plague bacillus, ray fungus, sarcinæ and moulds are the commoner micro-organisms found in pathological urine. The typhoid bacillus, which is eliminated by the kidney in every case of enteric fever, is occasionally the exciting factor of inflammatory disease of the bladder and renal pelvis.

Tubercle bacilli in the urine may indicate a tuberculous focus in the urinary tract. Their elimination by the kidneys from the blood in the absence of genito-urinary tuberculosis has been suggested.

Watson's method for the demonstration of tubercle bacilli in the urine is very satisfactory.[1] *Technic.*—Irrigate the glans penis and urethra with sterile water and have the patient void in three glasses. The last glass is a 250 c.c. conical shaped sedimenting glass which is capable of being fitted in a high powered laboratory centrifuge. If unable to secure the desired amount—200 c.c.—the specimen is set aside under sterile conditions and the process repeated as often as required. The urine may be secured by catheter and the entire quantity used.

Centrifuge the specimen for five minutes. If much sediment is present add 5 c.c. of antiformin and thoroughly mix. The specimen is again centrifuged—thirty to forty-five minutes—after which the supernatant fluid is decanted off and the sediment used for preparing glass slides. The slides are dried in air and fixed by heat. If the slides seem too thick, place in a

[1] Watson, Am. Jour. Med. Sc., Nov. 1918.

5 per cent. acid (HCl) alcohol mixture for two minutes. The slides are now submerged in a solution of carbol fuchsin for ten minutes. The solution is heated until it steams. Wash slides with water, place in a 2 per cent. acid (HCl) alcohol solution until completely decolorized, and counterstain with Loeffler's methylene blue.

The smegma bacillus which exists in the secretions of the external genitals is differentiated from the tubercle bacillus by the acid alcohol method or by Pappenheim's stain.

Yeast cells found in diabetic urine may give rise to pneumaturia.

Animal Parasites.—The Trichomonas vaginalis is rarely noted and its presence is probably dependent upon contamination of the urine with a vulvovaginal discharge containing this parasite. Ova of the Distoma hæmatobium are sometimes seen in the urine when the adult worm resides in the mucous membrane of the renal passages. Distomiasis is essentially a tropical disease which is occasionally responsible for hæmaturia. Filarial embryos have been found in certain cases of tropical hæmaturia. Echinococcus hooklets or fragments of cysts may be present in cases of hydatid disease of the urinary system. There are a few instances on record in which the Eustrongylus gigas was noted in the urine.

Calculi.—Urinary calculi of renal and vesical origin vary in size and outline. Stones small enough to pass through the urinary passages are more common than the large calculi found in the renal pelvis or bladder.

Uric acid stones vary in size from that of a grain of sand to concretions large enough to fill up the renal pelvis. These calculi are reddish-brown or dark gray, very dense, have a smooth or slightly roughened surface, dissolve in alkalies, and, when treated with sodium hydrate, generate ammonia. They give murexide test. Sometimes calcium oxalate is present in uric acid concretions. Ammonium urate stones have a waxy consistence, give the murexide test and liberate ammonia when treated with sodium hydrate. These calculi are rare. At times they are found in adults, and are occasionally discovered in the new-born. Calcium oxalate stones are responsible for severe attacks of renal colic and hæmaturia. These calculi are very hard, their surface is generally irregular, often showing sharp projections, and their color is dark gray or black. Hydrochloric acid dissolves them, and acetic acid will also cause solution when added to the powdered stone. Phosphate stones have a soft texture, are white or pale yellow, and have a rough surface. They are soluble in acetic acid without gas formation. They are formed in the bladder much more frequently than in the renal pelvis. Cystin stones have a wax-like consistency, are white or yellowish in color, dissolve in ammonia, and give the reaction for cystin. They are of rare occurrence. Xanthin stones are hard, of a white or yellowish-brown color, and dissolve in ammonia. Indigo stones have a blue or bluish-gray color. Xanthin and indigo calculi are extremely rare. Calcium carbonate stones are white, have a chalk-like consistency, and are soluble in acetic acid with gas formation. Stones consisting of fatty acids and cholesterin have been recorded in a few instances.

Tumor fragments from carcinoma or sarcoma of the urinary tract are rarely present in the urine. Fecal matter has been found in the urine in cases of enterovesical fistula.

CHEMICAL EXAMINATION.

Nitrogenous Bodies.—The normal amount of nitrogen eliminated by the kidneys per day varies between 10 and 16 grammes. It may be reduced to 5 or 6 grammes on a vegetable diet. Nitrogen, the best index of proteid metabolism, is principally eliminated in the form of urea, and to some extent as ammonia, uric acid, and extractives. Hammarsten's estimation of the percentage of nitrogen excreted in the principal nitrogenous bodies is as follows:

	Adults, per cent.	Infants, per cent.
Urea	84 to 91	73 to 76
NH_3	2 to 5	7.8 to 9.6
Uric acid	1 to 3	3 to 8.5
Extractives	7 to 12	7.3 to 14.7

Nitrogen is increased by a rich proteid diet, active exercise, in fevers, in cachexia, in diabetes, in poisoning by arsenic, antimony, phosphorus, and certain organic poisons, after hemorrhage, in dyspepsia, during the resolution stage of pneumonia, and from the absorption of exudates and transudates. It is lowered from lack of exercise, by a vegetable diet or one containing much carbohydrate, during the convalescence of fevers, in persons gaining weight rapidly, during pregnancy, during the formation of exudates and transudates, and in nephritis.

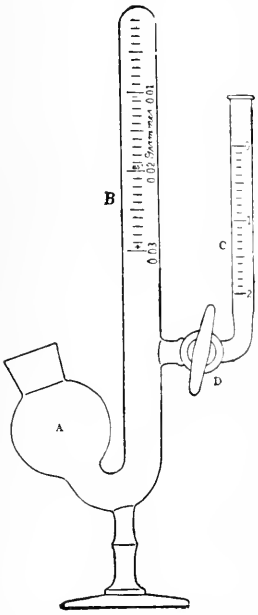


FIG. 119.—Heintz modification of Hüfner apparatus for urea determination. *A*, bulb; *B*, graduated tube to collect and measure the nitrogen; *C*, tube for urine; *D*, stop-cock.

Urea.—QUANTITATIVE ESTIMATION; HYPOBROMITE METHOD.—This quantitative test is based upon the principle that an alkaline solution of hypobromite of soda will decompose urea into nitrogen, and carbon dioxide, which is absorbed in the excess of alkali. The amount of urea is estimated by the volume of nitrogen set free. Hüfner has shown that one cubic centimetre of nitrogen (at 0° C. and 760 mm. pressure) represents .00268 gramme of urea. A convenient method (Rice) of preparing the hypobromite reagent is as follows: (1) A solution is made by dissolving 100 grammes of NaOH in 250 c.c. of water; (2) a solution of bromine one part, potassium bromide one part, and water eight parts. These solutions are mixed in equal amounts. Special forms of apparatus have been devised for collecting the nitrogen and measuring its volume. The Heintz modification of the Doremus apparatus can be highly recommended because it is easy to operate and is sufficiently accurate for clinical purposes. With this apparatus the test is conducted as

follows: The large tube is filled with hypobromite reagent and the small tube with urine up to the point indicated by the mark 1. By opening the stop-cock, one cubic centimetre of urine is allowed to flow very slowly into the large tube. The reaction occurs immediately and nitrogen gas is set free and collects in the upper part of the tube by displacing the fluid. The apparatus is then set aside for fifteen minutes, when the reading is taken. The amount of urea for one cubic centimetre of urine is indicated by a graduated scale at the upper level of the fluid. Albumin should always be removed before making this test.

The Urease Method for the determination of urea is probably the most satisfactory. The method depends upon the principle that the enzyme urease is able at ordinary temperatures to transform urea into ammonium carbonate, quickly and completely. The urine is treated with urease. Aërate the ammonia formed into fiftieth normal acid and titrate the excess acid with fiftieth normal alkali. The number of c.c. of fiftieth normal acid neutralized is multiplied by the factor 0.056 to measure the number of grammes of urea—plus ammonia-nitrogen in 100 c.c. of urine.[1]

On an ordinary diet the daily amount of urea varies between 20 and 40 grammes, on a rich diet it may reach 100 grammes, while on a restricted diet it is sometimes reduced to 15 grammes. As a rule the quantity of urea and the total nitrogen output are parallel, so that for clinical purposes the amount of urea is generally determined instead of the total nitrogen. Urea may show a reduction with a rise in the ammonia elimination.

Uric Acid.—Folin's Modification of Hopkin's Test.—Three hundred cubic centimetres of urine are treated with 75 c.c. of a reagent prepared as follows: 500 grammes of ammonium sulphate and 5 grammes of uranium acetate are dissolved in 650 c.c. of water, to which are added 60 c.c. of a 10 per cent. acetic acid solution, and water enough to bring the amount up to 1 litre. After standing for about five minutes the urine so treated is filtered through two thicknesses of filter-paper. Into each of two beakers 125 c.c. of filtrate are poured, treated with 5 c.c. of concentrated ammonia, and set aside for twenty-four hours. The ammonium urate precipitate is next washed with a small quantity of a 10 per cent. solution of ammonium sulphate. The precipitate of ammonium urate collected on filter-paper is washed with 100 c.c. of water into a beaker, after perforating the filter-paper. The solution is finally treated with 15 c.c. of concentrated sulphuric acid and then immediately titrated with a 1/20 normal solution of potassium permanganate, until a faint red color tints the entire solution. This color disappears rapidly. Each cubic centimetre of a 1/20 normal permanganate solution represents .00375 gramme of uric acid.

Uric acid is an oxidation product of the xanthin bases. Its origin depends upon the nucleins derived from the food (exogenous uric acid) and from the body tissues (endogenous uric acid). The normal daily amount of uric acid found in the urine varies between .2 and 1.25 grammes, which represents from 1 to 2 per cent. of the total nitrogen output. Uric acid is increased by a diet rich in nuclear proteids, active muscular exercise, in

[1] See Fiske (Journ. Biol. Chem., 23–455, 1915), Van Slyke and Cullen (Journ. Biol. Chem., 24, 117, 1916) and Hawk (Practical Physiological Chemistry, 5th edition) for details of method.

fevers, in anæmia, in leukæmia, in pneumonia during the stage of resolution, in cirrhosis of the liver, and in diabetes mellitus. In gout the amount of uric acid is generally decreased between the acute attacks, and rises during and immediately after the paroxysm. In gout an increase of uric acid is found in the blood (uratæmia). The mere existence of uratæmia does not justify the conclusion that it is the principal or primary factor of this disease; on the contrary, it would appear that an increase of urates which occurs in a number of conditions, as anæmia, leukæmia, and during the resolution stage of pneumonia, does not in itself favor precipitation of biurate of sodium. It has been suggested that an excess of sodium salts in the blood, lymph, and especially in synovial fluid, determines the precipitation of urates. Solutions of uric acid have been shown to possess only slightly toxic or harmless properties when injected into the tissues of animals.

The quantity of uric acid in the urine is decreased on a restricted diet, especially one poor in substances containing nucleins, after the administration of large doses of quinine, in nephritis, and in certain chronic diseases. At the present time a final opinion as to the rôle played by uric acid in the so-called uric acid diathesis cannot be given.

Xanthin Bases.—Under this heading is included a group of substances found in the urine in very small amounts and regarded as being formed from nucleins. In this group may be included xanthin, hypoxanthin, heteroxanthin, paraxanthin, guanin and adenin. In the main it may be said that the amounts of uric acid and the xanthin bases fluctuate in a parallel manner. The xanthin bases are increased in the urine in leukæmia, after a diet rich in nucleins, and in pneumonia. Rarely, calculi consist of xanthin.

Ammonia.—The normal daily output of ammonia is about 0.7 gramme, which represents slightly over four per cent. of the total nitrogen elimination. It exists in combination with some of the urinary acids. Its presence in the urine is accounted for by a small amount of ammonia which is not transformed into urea in the liver. Ammonia is increased in conditions associated with deficient oxidation, as cardiac dyspnœa, in certain diseases of the parenchyma of the liver, such as acute yellow atrophy and phosphorus poisoning, in diabetes mellitus, and, notably, in pernicious vomiting of pregnancy.

Chlorides.—Quantitative Determination.—Ten cubic centimetres of urine are diluted with 90 c.c. of water, to which are then added a few drops of a strong potassium chromate solution. A standard silver solution (1 c.c. of which represents .0035 gramme of chlorine, or .0058 gramme of NaCl) is then slowly added from a graduated burette. The development of a permanent orange color indicates that all the chlorine has been precipitated.

The excretion of chlorides, which varies from 10 to 15 grammes per day, depends almost exclusively upon the quantity of chlorides ingested. A decreased elimination is present on a diet poor in chlorides, in the acute fevers (probably due to a deficiency of chlorides in the fever diet), before the crisis in pneumonia, in acute and chronic nephritis, in many chronic diseases, in gastric disorders associated with vomiting, in diseases attended with diarrhœa, and during the formation of transudates and exudates. An augmented elimination is observed after a diet rich in chlorides, after

the acute fevers, especially during the stage of resolution of pneumonia, in diabetes insipidus, and from rapid resorption of transudates and exudates.

Phosphates.—Phosphoric acid of the urine is combined with sodium, potassium, ammonium, calcium, and magnesium. The daily amount of phosphoric acid excreted by the kidneys varies between two and three grammes. A diminished excretion has been noted in some febrile diseases, in cases of arthritis, between the paroxysms of gout, in pregnancy, in acute yellow atrophy of the liver, in nephritis, in Addison's disease, and in chronic lead poisoning. An increased elimination has been noted on a diet rich in meat, during the attack of gout, in diabetes mellitus, in neurasthenia, in hysteria, in leukæmia, and after active muscular exercise. The existence of a phosphatic deposit in the urine is not necessarily a sign of increased elimination, and is frequently due to alkalinity of the urine. A quantitative estimation of phosphoric acid is necessary to establish an increased output. Neubauer's method consists in titrating the urine with a uranium nitrate solution, using cochineal as an indicator. For the details of this method special works on urinary chemistry should be consulted.

Sulphates.—Sulphuric acid exists in the urine as mineral, preformed or neutral sulphates, and as conjugate or ethereal sulphates. The total daily output of sulphuric acid varies between 2 and 3 grammes, nine-tenths of which is eliminated as mineral sulphates and the remainder as ethereal sulphates. Ethereal sulphates occur in combination with certain aromatic bodies, the most important of these being phenol, indoxyl, skatoxyl, and cresol. The sulphate elimination is controlled principally by proteid metabolism, so that the amount is increased after a diet rich in meat, by muscular exercise, in the acute febrile diseases, in acute inflammatory diseases of the brain and spinal cord, and by certain poisons which augment proteid destruction. The output of sulphates is reduced by a vegetable diet or one poor in proteids, during the period of convalescence from the acute fevers, and in many chronic diseases. The quantity of ethereal sulphates depends mainly upon putrefactive changes occurring in the intestinal tract, and sometimes in other parts of the body. The normal proportion of ethereal sulphate to neutral sulphate varies considerably. The conjugate sulphates are diminished by starvation, by the administration of calomel and hydrochloric acid, and are increased by the ingestion of alkalies and carbolic acid, in intestinal diseases associated with increased putrefaction, as in constipation, enteric fever, and tuberculous enteritis.

Indican.—Obermayer's Test.—The reagent for this method is made by dissolving two parts of ferric chloride in 1000 parts of concentrated hydrochloric acid. A small amount of urine is treated with an equal part of Obermayer's reagent and the mixture shaken with 2 or 3 cubic centimetres of chloroform, which extracts indican. It is light blue or colorless when a normal amount is present, while an increased quantity is shown by a dark blue color.

Jaffe's Test Modified by Stokvis.—Equal volumes of hydrochloric acid and urine are mixed. The liquid is treated with a droplet of a concentrated solution of sodium or calcium hypochlorite and then shaken with a few c.c. of chloroform. A blue color is imparted to the chloroform by the

indigo. An approximate estimate of the amount may be formed by the depth of this color. Iodine in the urine tints the chloroform pink. Bile pigment should always be removed with lead subacetate before testing for indican. Indol is formed in the intestines as a result of putrefactive processes; in the blood it is oxidized and combines with sulphuric acid, being eliminated as sodium or potassium indoxyl sulphate or indican. As putrefaction is essential for the formation of indican, only small traces of this substance occur in the urine of healthy persons, since intestinal decomposition is slight under normal conditions. The quantity of indican is influenced by the character of food, being smaller upon a milk than on a full mixed diet. Jaffe found that 6.6 mg. was the average normal amount for 1000 c.c. of urine.

Pathological indicanuria occurs in carcinoma of the stomach, in certain forms of gastritis, and in conditions associated with inhibited intestinal peristalsis, as constipation, intestinal obstruction, and peritonitis. The amount of indican is augmented in putrid bronchitis, in empyema, and in gangrene and abscess of the lungs.

Urinary Pigments.—The color of normal urine depends chiefly upon urochrome. The following pigments are responsible for the color of many abnormal urines: pathological urobilin, uroerythrin, hæmoglobin, methæmoglobin, urohæmatin, uroroseinogen, biliary pigment, and melanin. After the ingestion of senna, santonin, iodine, phenol, and creosote abnormal pigmentation of the urine often occurs.

Biliary Pigments.—Rosenbach's Modification of Gmelin's Method.—The urine is filtered through thick filter-paper. A drop of concentrated nitric acid is then placed upon the urine-soaked filter-paper. A play of colors, consisting of red, yellow, green, blue and violet, in which the green predominates, will develop in the presence of biliary pigment.

Smith's Test.—A mall amount of tincture of iodine diluted with 10 parts of alcohol is added to 5 or 10 c.c. of urine, so that the iodine solution forms a layer above the urine. An emerald color forms at the zone of contact of two fluids when bilirubin is present. Biliary acids are associated with bilirubin so that their clinical significance is practically the same. The tests for biliary acids are attended with considerable difficulty.

The biliary pigments are bilirubin, biliverdin, bilifuscin, and biliprasin. Bilirubin is found in freshly voided urine only, while the other pigments may appear after the urine has stood for a time. Biliary pigment occurs in the urine in both toxæmic and obstructive jaundice.

Phenol.—Salkowski's Test.—About 10 c.c. of urine is treated with a few c.c. of nitric acid and boiled. On cooling, bromine water is added. An increased amount of phenol is shown by the development of a decided cloudiness or precipitate.

The amount of phenol eliminated is very small (0.3 gramme daily under normal conditions). This substance is increased whenever putrefactive processes occur in the body, as in gangrene, putrid bronchitis, empyema, and, rarely, from intestinal decomposition. It has also been demonstrated in tuberculosis, meningitis, peritonitis, erysipelas, scarlet fever, and from poisoning with phenol or some of its derivatives, such as salicylic

acid, pyrocatechin and hydroquinone. The urine containing phenol may become dark brown or black on standing.

Pathological Urobilin. — BRAUNSTEIN'S TEST.—About 20 c.c. of urine are mixed with 5 c.c. of a reagent which consists of 100 parts of a concentrated solution of cupric sulphate, 6 parts of hydrochloric acid, and 3 parts of ferric chloride. A small amount of chloroform is added to the mixture. On shaking, the chloroform becomes rose colored.

This pigment is closely related to urochrome and can be differentiated from the latter by the spectroscope. Urobilin and its chromogen are soluble in chloroform and precipitated with ammonium sulphate. Pathological urobilin is sometimes encountered in the urine in febrile diseases, cirrhosis of the liver, pernicious anæmia, cancer, cerebral hemorrhage, scurvy, Addison's disease, hæmophilia, and syphilis.

Melanin and Melanogen.—These substances are occasionally found in the urine of persons suffering from melanotic tumors, chronic malaria, and certain wasting diseases. The urine containing melanin and melanogen may have a normal yellow color when voided, but becomes darker when exposed to the air.

Albumins.—The proteids found in the urine are serum albumin, serum globulin, nucleo-albumin, albumose, Bence-Jones's albumin, hæmoglobin, fibrin and histon. The most important of these from a clinical standpoint is serum albumin.

Serum Albumin.—The most useful tests for the detection of albumin are the boiling and acidulation tests and Heller's test, because they afford uniformly satisfactory results, are simple and easily applied. It is claimed that these tests are less sensitive than many others, such as Speigler's and Tanret's. Before testing for albumin the urine should be clear, and, if cloudy, must be filtered through several layers of filter-paper. Bacteria cannot be completely removed by filtration through ordinary filter-paper. It is desirable to have a fresh specimen for testing. In certain cases several samples should be secured, *i.e.*, the first urine passed in the morning on arising, and that voided late in the afternoon. Albumin reactions are sometimes less distinct in concentrated specimens than in those of low specific gravity, and it is, therefore, advisable to dilute an inspissated urine before applying albumin tests.

BOILING AND ACIDULATION TEST.—Clear urine is boiled in a test-tube. When a precipitate forms this is generally due to either phosphates or albumin (serum albumin in conjunction with serum globulin). The turbidity caused by phosphates clears on the addition of a few drops of colorless nitric acid, while the cloud due to albumin remains or even is intensified after acidulation. A precipitate of carbonates, developing on heating, will disappear upon the addition of nitric acid with the liberation of gas (CO_2). If on boiling the urine remains clear but subsequently on cooling a cloud develops, this is due to albumose. This turbidity will again disappear on heating. Certain resinous bodies, as copaiba, benzoin, cubebs, and turpentine, also produce a precipitate on heating. This cloud can be distinguished from that produced by albumin by the fact that alcohol dissolves the turbidity produced by these substances. When employing

acetic acid, it is best to add a few drops before boiling, care being taken to avoid an excess, since albumin may not precipitate on boiling. If a cloud forms after the urine is treated with acetic acid, this is caused by nucleo-albumin and should be removed by filtration before testing for serum albumin. The most accurate results are obtained with this method when a dilute acetic acid solution is employed (25 per cent.).

Heller's Test.—Colorless nitric acid is allowed to flow slowly from a pipette into a test-tube or a conical glass vessel containing a small quantity of urine, so that the urine forms a distinct layer above the acid. In order to prevent mixing the acid and urine, the test-tube or conical vessel should be inclined while adding the nitric acid. When serum albumin is present a white disk appears at the zone of contact between the urine and acid. When a small amount of albumin exists the precipitate does not form immediately but in the course of several minutes. An approximate quantitative estimate of albumin can be formed from the thickness of the coagulated layer. A pale red or reddish-violet disk, at or above the plane of contact, is noted in many normal and abnormal urines. A white precipitate is also caused by serum globulin and albumose. The latter disappears on heating and reappears on cooling. Nucleo-albumin in large amounts may give a positive reaction, but this is so uncommon that it can be disregarded for practical purposes. Certain resinous bodies, indicated in the discussion of the boiling and acidulation test, produce a white cloud which disappears when treated with alcohol.

Acetic Acid and Potassium Ferrocyanide Test. — A few drops of 10 per cent. solution of potassium ferrocyanide or platinocyanide are added to a small amount of urine previously acidified with acetic acid. A precipitate indicates albumin or albuminose. If, on heating, the turbidity disappears completely, the presence of the latter substance is indicated, or, if the cloud partly clears on warming, the presence of both substances may be inferred. When a precipitate, due to nucleo-albumin, forms on addition of acetic acid, the urine should be filtered and the test repeated.

Tanret's Test.—The reagent is prepared as follows: Dissolve 1.35 grammes of mercuric chloride in 25 c.c. of water, add to this solution 3.32 grammes of potassium iodide dissolved in 25 c.c. of water, then make the total solution up to 60 c.c. with water and add 20 c.c. of glacial acetic acid to the combined solutions.

Technic.—To 5 c.c. of albumin solution in a test-tube add Tanret's reagent, drop by drop, until a turbidity or precipitate forms. This is an exceedingly delicate test. Sometimes the albumin solution is stratified upon the reagent as in Heller's or Roberts' ring tests. It is claimed by Repiton that the presence of urates lowers the delicacy of the test. Tanret claims that the removal of urates is not necessary inasmuch as the urate precipitate will disappear on warming and the albumin precipitate will not. He says, however, that mucin interferes with the delicacy of his test and should be removed by acidification with acetic acid and filtration before testing for albumin.

Spiegler's Test.—The test solution as modified by Jolles consists of mercuric chloride 10 grammes, succinic acid 20 grammes, sodium chloride

20 grammes, and distilled water 500 c.c. The reagent is added slowly by means of a pipette to a small amount of urine contained in a test-tube, so that the urine forms a layer above the test solution. A white cloud at the junction of the fluids indicates albumin, nucleo-albumin or albumose. When the urine contains iodine, a precipitate of mercuric iodide forms, which is soluble in alcohol. This test is very sensitive.

Many other methods for the detection of albumin are recommended by different authorities, as tests with picric acid, metaphosphoric acid, phosphotungstic acid, and trichloracetic acid.

Quantitative Determination of Albumin. Esbach's Method.—The test solution is prepared by dissolving 10 grammes of picric acid and 20 grammes of citric acid in 1000 c.c. of distilled water. A special graduated test-tube devised by Esbach and known as an albuminometer is required for this method. The urine should have an acid reaction. It is poured into the albuminometer to the mark "U"; the reagent is then added until the fluid reaches to the mark "R." The fluids are then mixed and the test-tube set aside for twenty-four hours, when the reading is taken. The height of the column of coagulated albumin, as measured by the scale on the tube, represents the amount pro mille. Esbach's reagent precipitates serum albumin, serum globulin, albumose, uric acid, and creatinin. When the specific gravity exceeds 1.008, or when a large amount of albumin exists, the urine should be diluted with one or several volumes of water before applying the test. The reading is multiplied by the number of dilutions. Esbach's method, although not so accurate as the gravimetric determination, is quite satisfactory for general clinical purposes.

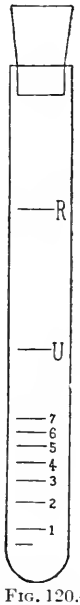


Fig. 120.—Esbach's albuminometer. — Emerson.

Boiling Test.—An approximate estimate of the quantity of albumin can be formed by boiling acidified urine in a test-tube and allowing the precipitate to settle for twenty-four hours. The error with this method may be considerable, because albumin sometimes separates in large and at other times in small flakes.

Gravimetric Method.—One hundred cubic centimetres of urine are sufficiently acidulated with acetic acid to insure separation of all the albumin. It is then boiled and passed through a filter of known weight. The precipitate collected on the filter is washed with hot water until the washings cease to give a reaction for chlorides. The precipitate is next washed successively with alcohol and ether to remove fat. The filter containing the precipitate is now dried at a temperature of 120° to 130° and then carefully weighed. The weight of the albumin is obtained by subtracting the weight of the filter-paper from the combined weight of the filter-paper and dried precipitate.

Albuminuria.—The term albuminuria implies the presence in the urine of coagulable albumin, and refers particularly to serum albumin. One or more albuminous bodies are almost invariably associated with serum albumin. Albuminuria is symptomatic of a large number of morbid states, from minor disturbances in health to malignant diseases.

1. Renal Albuminuria.—When albumin is eliminated by the kidneys the condition is termed renal albuminuria.

(a) *So-called physiological albuminuria* is occasionally noted in healthy individuals after violent exercise or severe nervous stress. Whether albuminuria is ever physiological is still a mooted question. Albuminuria often occurs in pregnancy, especially in the later stages. The so-called albuminuria of adolescents is probably pathological.

(b) *Albuminuria of Organic Kidney Disease.*—In this variety the presence of albumin in the urine depends directly on structural changes in the renal tissues, and in nephritis, and amyloid, tuberculous, malignant and cystic disease of the kidney. In acute and chronic parenchymatous nephritis the amount is generally large, while in amyloid disease it is moderate or small, and in contracted kidney it is small. The mere presence of albumin in the urine never warrants a diagnosis of organic renal disease; on the other hand, mere traces occur in granular kidney, and, indeed, albumin may be absent for a time in this disease. Large quantities of albumin usually justify a diagnosis of organic kidney disease.

(c) *Febrile Albuminuria.*—A discharge of albumin of slight or moderate degree in fevers and inflammatory diseases is suggestive of a simple parenchymatous degeneration of the kidney and of vascular derangements, incident to the febrile or inflammatory process, while a high grade of albuminuria, noted in a limited number of these cases, points to marked renal degeneration, often associated with decided congestion. The difference between albuminuria of febrile and inflammatory disorders and that of acute Bright's disease is essentially one of degree, so that a sharp distinction cannot be made between these forms. Albuminuria is symptomatic of many of the infectious diseases, especially enteric fever, typhus fever, pneumonia, cerebrospinal fever, yellow fever, plague, cholera, malignant endocarditis, diphtheria, erysipelas, and variola.

(d) *Toxic Albuminuria.*—Under this heading is included the albuminuria produced by drugs, such as salicylic acid, potassium iodide, salol, urotropine, phenol, alcohol, ether, chloroform, lead, mercury, phosphorus, and a number of other toxic substances.

(e) *Albuminuria occurring in blood disorders* is seen in severe secondary anæmias, pernicious anæmia, chlorosis, and leukæmia.

(f) *Alimentary Albuminuria.*—The ingestion of very large amounts of albumin, such as raw eggs, may excite albuminuria, but a moderate quantity of albuminous food will never produce albuminuria in a healthy person. An antecedent chronic albuminuria may be intensified by a moderate consumption of albumin.

(g) *Albuminuria dependent upon circulatory disturbances of the kidneys* is seen in cardiac disease, especially during the stage of ruptured compensation, in pulmonary disease with venous stasis, from pressure on the renal veins by a tumor, cyst or peritoneal effusion, and by a thrombus in these vessels. In floating kidney albuminuria sometimes depends on kinking of the renal veins so that it may be present only while the individual is in the erect posture, disappearing when in the recumbent position (orthostatic albuminuria).

(h) *Albuminuria in nervous diseases* is common when organic lesions of the nervous system exist, such as apoplexy, brain tumor, and spinal sclerosis, but it is infrequent in functional disorders, such as neurasthenia and migraine.

(i) *Albuminuria caused by obstruction in the urinary passages* occurs in nephrolithiasis, when the stone blocks up the ureter for a time, and also when the ureter is compressed by a tumor or is twisted. The urine which has been impeded in its passage shows albumin in many instances.

2. Accidental Albuminuria.—When the urine contains albumin derived from the renal passages or genital organs it is designated accidental or extrarenal albuminuria. The presence of pus, blood, leucorrhœal discharge, and chyle in the urine, as a rule, causes a slight, and rarely, a moderate albumin reaction. This type occurs in pyelitis, ureteritis, cystitis, prostatitis, vesiculitis, epididymitis, urethritis, vulvovaginitis, and during menstruation. A vaginal discharge is often washed into the urine. The diagnosis of accidental albuminuria is generally unattended with difficulty, provided the results of microscopic examination and the clinical investigation are carefully considered. In general terms it may be said that the intensity of the albumin reaction is directly proportionate to the amount of cellular deposit. The differentiation between renal and extrarenal albuminuria rests on the data obtained by a careful urinalysis with other clinical findings. Both conditions often coexist. The presence of tube-casts and many pus-cells with an albumin reaction greater than the number of leucocytes would indicate, argues in favor of a coexistent renal and accidental albuminuria.

Serum Globulin.—Kauder's Test.—The urine is treated with a sufficient quantity of ammonia to separate the phosphates, which are removed by filtration. An equal bulk of a saturated solution of ammonium sulphate and filtrate are mixed. A precipitate represents serum globulin.

Serum globulin and serum albumin are almost invariably associated, so that their clinical significance is similar. As a rule serum albumin is found in excess of serum globulin, although exceptions to this rule are recorded in amyloid disease, diabetes, and severe nephritis.

Nucleo-albumin.—This body is precipitated by strong acetic acid. Concentrated urines should always be diluted with two or three volumes of water before applying this test. Urine containing much serum albumin and serum globulin should be boiled and filtered in order to remove these substances before testing for nucleo-albumin.

Ott's Method.—Add to the urine an equal volume of saturated solution of sodium chloride, and treat the mixture with Almen's tannin solution. The presence of nucleo-albumin is shown by the formation of an abundant precipitate. Almen's solution consists of 5 grammes of tannic acid, 10 c.c. of a 25 per cent. solution of acetic acid, and 240 c.c. of 50 per cent. ethyl alcohol. Nucleo-albumin can be removed from the urine with neutral lead acetate.

With certain delicate tests nucleo-albumin can be demonstrated in many normal and abnormal specimens, so that its presence in small amount may be regarded as physiological. When nucleo-albumin can be detected

by tests generally employed in routine clinical work, it is probably pathological. Nucleo-albuminuria occurs in inflammatory diseases, especially of a catarrhal nature, of the urinary tract, as cystitis and pyelitis. In febrile diseases associated with albuminuria, in leukæmia, in jaundice, and in acute nephritis, nucleo-albuminuria is not uncommon. In the last named disease nucleo-albuminuria sometimes precedes and follows serum albuminuria.

Albumose.—To the urine strongly acidulated with acetic acid, is added an equal amount of a saturated solution of sodium chloride. The presence of a precipitate, which disappears on boiling and returns on cooling the urine, consists of albumose. When serum albumin coexists with albumose, this must be removed by boiling and filtering before applying the test.

Albumosuria is referred to by some writers as peptonuria, a term which Kühne restricts to the presence of true peptone. According to Kühne, peptonuria has been found in pneumonia, phthisis, and gastric ulcer. The chief clinical significance of albumose in the urine relates to morbid lesions, characterized by a destruction of leucocytes, with the absorption of the disintegrated products. In many diseases showing these pathological features, especially in purulent collections, the occurrence of albumosuria may be a useful sign in diagnosis. In this connection it must be pointed out that, since the group of conditions in which it occurs is a vast one, its significance is of less value in diagnosis than any other urinary findings. Albumosuria has been noted in pneumonia during the period of resolution, in suppurative meningitis, in liver abscess, in septicæmia, in leukæmia, in endocarditis, in myxœdema, in diphtheria, in measles, in rheumatic fever, in scarlet fever, in acute yellow atrophy of the liver, in scurvy, in dermatitis, and in intestinal diseases characterized by ulceration, as enteric fever, tuberculosis, and carcinoma. Albumosuria may be associated with, or occur independently of, serum albuminuria.

Bence-Jones's Albumose.—The recognition of this proteid depends upon the fact that its precipitation occurs at a temperature of 52° to 60° C. Upon boiling, the cloud entirely or partially disappears, to return again on cooling. With Heller's nitric acid test Bence-Jones's albumin gives a reaction like that of serum albumin.

This proteid, first described by Bence-Jones, occurs with considerable frequency in myelomatous growths in the bones. It is generally designated as albumose, but probably incorrectly. The researches of Simon and Magnus Levy indicate that it is a true albumin.

Hæmoglobin.—The spectroscopic examination, as a rule, shows absorption bands of methæmoglobin, sometimes of oxyhæmoglobin.

Donogany's Test.—If, on the addition of 1 c.c. of ammonium sulphide solution and an equal quantity of pyridine to 10 c.c. of urine, an orange color develops, the presence of blood may be inferred. When the result is doubtful, a spectroscopic examination should be made of the mixture.

The physiological destruction of red corpuscles is not followed by hæmoglobinuria, because the coloring matter set free from the disintegrated erythrocytes is converted wholly, or in part, in the liver into bile, and, perhaps, a fraction of the amount is redeposited in the tissues and stored there for the future demands of the system. The explanation generally

offered to elucidate hæmoglobinuria is based upon an erythroeytolysis so excessive that a part of the hæmoglobin liberated into the plasma (hæmoglobinæmia) is secreted by the kidneys. Hæmoglobinuria occurs in some cases of malarial fever (black water fever). It has been observed in yellow fever, variola, icterus gravis, scarlet fever, enteric fever, syphilis, Raynaud's disease, and from the toxic action of phenol, potassium chlorate, snake venom, hydrogen sulphide, carbon monoxide, and after exposure to the cold. The etiological factor responsible for paroxysmal hæmoglobinuria has not been definitely determined. Some writers claim that exposure to cold is the exciting cause, while others hold that it is of nervous origin. Hæmaturia is much more common than hæmoglobinuria.

Fibrin.—The suspected fibrin clots are separated from the urine by filtration, then thoroughly washed with water and dissolved by boiling in a 5 per cent. solution of hydrochloric acid. The solution thus secured gives the test for serum albumin when the coagulum consists of fibrin.

Fibrinuria has been noted in hæmaturia, chyluria, and in pseudomembranous inflammation of the urinary tract.

An acetosoluble albumin referred to by Simon as Patein's albumin has been reported in cystic kidney and nephritis.

Glucose.—Fresh urine is desirable for quantitative examinations for sugar. When albumin is present, this should be removed from the urine before testing for glucose.

Fehling's Test.—Two solutions are required, an alkaline and a copper solution, which should be mixed just before applying the test. Fehling's reagent deteriorates in a few days to such an extent that it is unsuited for testing; therefore, it is necessary to keep the alkaline and copper solutions in separate bottles supplied with well-fitting rubber corks. The alkaline solution consists of potassium and sodium tartrate 173 grammes. potassium hydrate 60 grammes, and 500 c.c. of distilled water. The copper solution consists of cupric sulphate 34.64 grammes, dissolved in 500 c.c. of distilled water. Equal volumes of these solutions are poured into a test-tube and shaken; the mixture is then diluted with four parts of water and boiled. After removing the test-tube from the flame the urine is added in small amounts, and after each addition the mixture heated but not boiled. When sugar is present a yellow or red precipitate of cupric suboxide separates. A change of the blue color of Fehling's solution to green, with a slight turbidity of the liquid after the addition of the urine, is very often seen, and may not be caused by glucose. Nearly every reducing substance except sugar requires boiling to produce precipitation of cupric suboxide.

BENEDICT'S TEST—FORMULA

Copper sulphate (pure crystallized)	17.3 gm.
Sodium or potassium citrate	173.0 gm.
Sodium carbonate (crystallized)	200.0 gm.
or the anhydrous salt	100.0 gm.
Distilled water to make	1000.0 c.c.

The citrate and carbonate are dissolved together with the aid of heat in about 700 c.c. of water. The mixture is then poured, through a filter if necessary, into a larger beaker or casserole. The copper sulphate dissolved

separately in about 100 c.c. of water is poured slowly into the first solution, with constant stirring. The mixture is cooled and diluted to one litre. This test solution keeps indefinitely.

Technic.—For the detection of glucose in the urine about 5 c.c. of the reagent are placed in a test-tube and 8 to 10 drops of the urine to be examined are added. The mixture is vigorously boiled for one or two minutes, and allowed to cool spontaneously. In the presence of glucose the entire body of the solution will be filled with a precipitate, which may be red, yellow, or greenish in tinge. If the quantity of glucose be under 0.3 per cent. the precipitate forms only on cooling. If no sugar be present, the solution either remains perfectly clear, or shows a faint turbidity that is blue in color, and consists of precipitated urates. In the use of the reagent the addition of a small quantity of urine—8 to 10 drops—to 5 c.c. is important, not because larger amounts of normal urine would cause reduction of the reagent, but because more delicate results are obtained by this procedure. Vigorous boiling of the solution after addition of the urine, and allowing the mixture to cool spontaneously are also important. If sugar is present the solution, either before or after cooling, will be filled from top to bottom with a precipitate, so that the mixture becomes opaque. (Joslin.)

Phenylhydrazine Test.—About .5 gramme of phenylhydrazine hydrochloride and 1 gramme of sodium acetate are added to about 8 c.c. of urine contained in a test-tube. If the salts do not dissolve on warming the urine, water is added to effect solution. The tube is now placed in boiling water for 20 or 30 minutes, then removed, and rapidly cooled by placing the test-tube in cold water. The formation of a bright yellow precipitate indicates the presence of sugar. Mere traces of glucose cause a small amount of precipitate which should be examined microscopically for phenylglucosazone crystals. These consist of yellow needles arranged singly or in clusters. Their melting point is 205° C. In experienced hands this test is generally considered the most sensitive.

Nylander's Modification of Boettger's Test.—Almen's reagent, required for this method, consists of 4 grammes of potassium and sodium tartrate, 2 grammes of bismuth subnitrate, and 10 grammes of sodium hydrate dissolved in 90 c.c. of water. This solution is then boiled and, after cooling, is filtered. A small quantity of Almen's reagent is added to the urine, approximately in the proportion of 1 to 11, and the resultant mixture is boiled. In the presence of sugar a dark gray or black precipitate of metallic bismuth separates. A positive reaction may be given by albumin, melanin, melanogen, and other reducing substances found in the urine after the ingestion of salol, benzol, sulphonal, trional, turpentine, quinine, rhubarb, and senna.

Fermentation Test.—The principle of this method is based on the fact that glucose is decomposed by yeast into alcohol and carbon dioxide. Special fermentation tubes, as designed by Einhorn, are convenient in conducting this test. The method is carried out by mixing a bit of a cake of compressed yeast with urine in a test-tube. Einhorn's fermentation tube is filled with this mixture, care being taken to exclude air bubbles from

the top of the tube. The saccharometer is kept at a temperature of from 25° to 38° for twenty-four hours, during which time the CO_2 collects in the upper part of the tube. A temperature of 34° C. gives the most satisfactory results. A control test should always be made with normal urine, since slight fermentation occurs in every specimen. With Einhorn's tube, an approximate estimate of the quantity of sugar can be formed, but for accurate quantitative analysis Robert's differential method is to be preferred. The fermentation test serves to differentiate fermentable sugar from other reducing substances.

Quantitative Estimation of Sugar. Fehling's Titration Method.—Ten c.c. of Fehling's solution diluted with 40 c.c. of water are boiled. At this temperature the saccharine urine is added drop by drop from a graduated burette, until the blue color of the test solution disappears, which indicates complete reduction of cupric oxide. The presence of reduced copper held in suspension obscures the color of the solution, so that it is necessary to allow the cuprous oxide granules to settle from time to time in order to detect the tint of the fluid. The cupric oxide contained in 10 c.c. of Fehling's solution is reduced by .05 gramme of glucose.

Benedict's Method.—The estimation is based upon the fact that a given quantity of glucose will reduce a fixed amount of copper if the two are combined in an alkaline solution. The solution is decolorized and the copper precipitated as a snow-white compound—cuprous sulphocyanate.

The reagent is prepared as follows:

(1) Dissolve 18 grains of pure crystallized copper sulphate in 100 c.c. of water.

(2) Dissolve by the aid of heat, 200 grammes of sodium citrate, 200 grammes of crystallized sodium carbonate and 125 grammes of potassium sulphocyanide in 800 c.c. of water.

(3) Mix solutions one (1) and two (2), add 5 c.c. of a 5 per cent. solution of potassium ferrocyanide, cool, and dilute to exactly one litre. The solution keeps indefinitely.

Technic.—(1) Dilute 10 c.c. of urine with water to 100 c.c. and pour into a burette to the zero mark.

(2) Place 25 c.c. of the reagent in a porcelain dish, add 10 to 20 grains of sodium carbonate (crystallized) and a small quantity of talcum or pumice. Heat the mixture until the carbonate is dissolved.

(3) The copper solution (2) is briskly boiled and the diluted urine in the burette is run into the boiling solution until the color has entirely disappeared. This marks the end point. If the solution becomes too concentrated a small amount of water may be added.

The twenty-five cubic centimetres of copper solution are reduced by 50 mg. (0.050 gms.) of glucose.

The percentage of urine is determined as follows: $\frac{0.050}{X} \times 1000 =$ percentage in original sample, wherein x is the number of c.c. of the diluted urine required to reduce 25 c.c. of the copper solution. The daily quantity of the urine multiplied by percentage gives the number of sugar grammes for the day.

Robert's Differential Density Method.—The principle of this

method rests on determining the specific gravity before and after fermentation; each .001 degree of difference in the specific gravity represents .23 per cent. of sugar. The test is carried out by noting the specific gravity of 200 c.c. of urine taken from a mixed twenty-four-hour specimen. A portion of a cake of compressed yeast is mixed with the urine, which is then set aside for twenty-four or forty-eight hours. The glucose generally disappears in twenty-four hours, but, in order to ascertain whether all the sugar has been decomposed, the urine is tested by Fehling's method. After all the sugar has been decomposed, the specific gravity of the fermented urine is taken and the difference between the two readings determined. The small urinometers employed in clinical work are not suited for exact determination, therefore it is convenient to use larger instruments. Accurate estimations can be taken with a set of four or five hydrometers, each of which represents a part of the specific gravity range ordinarily encountered in diabetic urine. For example, hydrometer number 1 indicates the scale from 1.000 to 1.010; number 2 ranges from 1.010 to 1.020; number 3 ranges from 1.020 to 1.030; number 4 ranges from 1.030 to 1.040; number 5 ranges from 1.040 to 1.050. The specific gravity observations should be taken at, or nearly, the same temperature. Evaporation of the urine should be reduced to a minimum during fermentation. The first specific gravity determination is taken before the yeast is added to the urine, and the second reading is made after the fermented urine has been filtered.

The quantitative determination for sugar by the polariscope is recommended highly by many workers. A polariscope designed for this estimation is an expensive instrument. The rapidity with which a determination can be made is one of its chief advantages over other methods.

Physiological Glycosuria.—The presence of traces (.5 pro mille) of glucose in the urine of healthy persons is conceded by most authorities. This quantity cannot, however, be detected by the tests employed in routine work.

Pathological Glycosuria.—This condition may be said to exist when glucose can be recognized by the tests generally in vogue in clinical work. Glycosuria may be transitory, intermittent, or constant. The latter variety is one of the cardinal symptoms of diabetes mellitus.

Glycosuria depends directly on an excess of sugar (above .2 per cent.) in the blood. A possible exception to this rule relates to the glycosuria following the administration of phloridzin. It is thought that this substance produces such alterations in the renal epithelium as to permit of increased glucose elimination. A renal form of diabetes has been suggested. The sugar of the blood is derived principally from the carbohydrates of the food, and in all likelihood some glucose is produced from the albumins of the food. In certain cases of diabetes, characterized by rapid emaciation, body proteids are concerned in its formation. Although many factors involved in the physiology of glucose metabolism remain unexplained, much clinical and experimental evidence supports the view, (1) that sugar metabolism is to a great extent regulated by the nervous system, (2) that the liver is chiefly concerned in converting sugar into glycogen, and also in forming glucose, and (3) that the pancreas secretes a sugar-destroying ferment. A

hypothetical conception of pathological glycosuria based on this theory may be said to depend on a failure on the part of the liver to form and store up glycogen, a disturbance which might result from a loss of nervous control or from disease of the hepatic cells; or on an inability on the part of the system to consume sugar, which is ascribed to a disturbance in the function of the pancreas inhibiting or suppressing the secretion of the glycolytic substance. Clinically, glycosuria occurs under a variety of circumstances: Disorders of the nervous system. Temporary or permanent glycosuria is observed in brain tumors, meningitis, injuries to the nervous system, neurasthenia, exophthalmic goitre, and may follow worry, fright, or mental overwork. Diseases of the pancreas. Permanent glycosuria is often associated with selerosis, and sometimes with atrophy or tumors of the pancreas, while temporary glycosuria is at times symptomatic of acute inflammation of this organ. Hepatic disease, abscess and cirrhosis of the liver may be attended with the temporary or constant presence of sugar in the urine. Toxic agents. The occasional occurrence of glucose in the urine is noted in the infectious diseases, as syphilis, influenza, enteric fever, diphtheria, rheumatic fever, and malaria, and from poisoning by chloral, alcohol, and morphine. The explanation of glycosuria occurring under these circumstances might be found in the development of a disorder of the function of the liver, the pancreas, or the nervous system, produced by these toxic agents. This variety is mainly observed as a transitory form, although occasionally diabetes develops after an acute infectious disease, which suggests permanent morbid processes of the hepatic or pancreatic tissues excited during the acute stage of the disease.

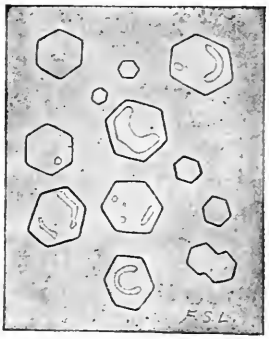


Fig. 121.—Iodoform crystals formed from the distillate of the urine of a case of diabetes. —Emerson.

The power possessed by the system to consume sugar varies in health and in disease. Carbohydrate tolerance can be determined by the administration of glucose by the mouth. The urine of healthy persons generally does not show glucose unless the amount ingested exceeds 250 grammes. When glycosuria follows the taking of 100 grammes, an abnormal sugar metabolism probably exists (pathological alimentary glycosuria). Carbohydrate tolerance is lessened by age, and is often reduced in obesity and gout.

Lactose.—The presence of milk sugar in the urine is indicated by a positive reaction with Trommer's and Nylander's tests after prolonged boiling, when negative results are obtained with the phenylhydrazine and fermentation tests. Lactose is found in the urine during the last weeks of pregnancy and in nursing women. Glycosuria and lactosuria are occasionally associated. The ingestion of more than 120 grammes of lactose often causes a lactosuria.

Levulose.—The presence of fruit sugar may be inferred when the urine gives sugar reactions with Trommer's, Fehling's, the fermentation and phenylhydrazine tests, and does not rotate polarized light to the right. Levulose at times rotates polarized light to the left. Levulose occurs in

the urine in some cases of diabetes and, at times, in the urine of healthy persons after the ingestion of levulose.

Pentose.—Pentose can be recognized by the fact that it does not undergo fermentation with yeast, but gives a positive reaction with Fehling's, Nylander's, and the phenylhydrazine tests. Pentose has been discovered in the urine after eating plums, pears, apples, cherries, and huckleberries, from the ingestion of 50 grammes or more of pentose, and occasionally in diabetes. A family tendency has been recorded.

Dextrin.—This substance reduces Fehling's solution, the copper separating first as a green, then changing to a yellow precipitate, and sometimes as a dark brown sediment. Dextrin has been found in the urine in the absence of glucose. Some authorities regard the presence of traces of dextrin as normal.

Acetone.—Legal's Test.—A few drops of freshly prepared concentrated solution of sodium nitroprusside are added to a small amount of urinary distillate, and the mixture treated with sodium or potassium hydrate. When a ruby color develops, rapidly changing to yellow, it signifies the presence of acetone. This test is usually negative with mere traces of acetone.

Lieben's Test.—A few drops of potassium hydrate solution and a small quantity of iodopotassic iodide are added to the urinary distillate, and the mixture warmed. Acetone is indicated by the formation of iodoform, which appears as hexagonal or stellate crystals, and can be recognized by its characteristic odor.

Gunning's Test.—Tincture of iodine, or Lugol's solution, is added to the urinary distillate, and the mixture treated with ammonia until a black precipitate develops, which slowly disappears, leaving a yellow deposit of iodoform crystals.

Acetone occurs in normal urine in small quantities, not exceeding 10 mg. in twenty-four hours. It is increased by restricting or withholding carbohydrates from the diet, especially when large amounts of proteids are consumed. It is also augmented in febrile diseases, in certain cachexias, in gastric ulcer, and follows the administration of phloridzin, and chloroform narcosis, and in severe forms of diabetes mellitus, notably before and during diabetic coma.

Diacetic or Aceto-acetic Acid.—Gerhardt's Test.—Ten or 15 c.c. of urine are subjected to the action of a solution of ferric chloride. When a precipitate forms on the addition of the ferric chloride, it is removed by filtration, and to filtrate is again added the test solution. Diacetic acid may be inferred when a Bordeaux red color develops, which may completely disappear in from twenty-four to forty-eight hours. Salicylic acid, salol, aspirin, diuretin, sodium acetate, and antipyrin may give a similar reaction. Prolonged boiling of the urine containing diacetic acid will cause a complete or partial disappearance of this substance.

Diacetic acid is rarely found in normal urine. It occurs in conjunction with large amounts of acetone, and the clinical significance of aceto-acetic acid is similar to that of acetone. Oxybutyric acid may also be associated with diacetic acid Diaceturia is of special importance in diabetics, since

it is a trustworthy sign of acidosis, and is always a forerunner of diabetic coma. Aceto-acetic acid has been noted in the urine in febrile diseases, in gastro-intestinal disturbances, especially those attended with starvation, and occasionally in individuals who have consumed a rich proteid diet for a number of days.

β-Oxybutyric Acid.—The urine is evaporated to the consistency of a syrup, and an equal volume of concentrated sulphuric acid is added. By distillation crotonic acid is obtained. Crystals of crotonic acid separate on cooling the distillate. If crystallization does not occur readily, an ethereal extract is obtained, evaporated, and the residue dissolved in water and allowed to crystallize. The presence of β-oxybutyric acid may be inferred by these crystals. If fermented diabetic urine containing oxybutyric acid be subjected to polariscopic examination, polarized light is rotated to the left.

β-Oxybutyric acid is the mother substance of diacetic acid, while acetone is derived from the latter substance. Its presence may be suspected when diacetic acid exists in the urine in large amounts. β-oxybutyric acid occurs less frequently than diacetic acid and acetone, and in general terms may be said to arise under conditions similar to those causing acetonuria. It is found in the urine in severe infectious fevers, during starvation, and in grave forms of diabetes. β-oxybutyric acid is generally regarded as the cause of diabetic coma. Some attribute the symptoms of this condition to a lowering of the alkalinity of the blood (alkali starvation), others contend that its toxic action is responsible.

Alkaptone Bodies.—The urine containing alkaptone bodies reduces Fehling's reagent, causing this test solution to blacken. This reaction serves to differentiate it from glucose. Nylander's, the phenylhydrazine and the fermentation tests are negative with urine containing alkaptone bodies.

Urine of alkaptonuric individuals appears normal when voided, but on standing its color changes to a reddish-brown or black. This peculiar characteristic of the urine is thought to be due to homogentisinic acid and uroleucinic acid. The cause of this condition is not known. The condition is compatible with good health, and is often peculiar to several members of a family, but inheritance does not seem to be an important factor in its production.

Ehrlich's Diazo Reaction.—This test, introduced by Ehrlich, depends on certain diazo bodies, which probably combine with aromatic compounds, giving a color reaction. The test is conducted as follows: A solution consisting of 5 parts of sulphanilic acid, 50 parts of hydrochloric acid, and 1000 parts of water, is mixed with a .5 per cent. solution of sodium nitrite in the proportion of 50 of the former to 1 of the latter. An equal volume of urine is added to this mixture and shaken. Upon the addition of a few drops of ammonia, a cherry-red color develops at the zone of contact, indicating a positive diazo reaction. On shaking, the entire fluid becomes red. A brown or salmon color constitutes a negative reaction. The chief clinical significance of this reaction relates to its almost constant presence in enteric fever, but is without value as a differential sign, since it occurs in a number of diseases. It is frequently present in measles, and occasionally

in pneumonia, scarlet fever, diphtheria, phthisis, rheumatic fever, meningitis, and at times in non-febrile diseases, such as chronic nephritis, carcinoma of the stomach, and leukæmia. The administration of salol, phenol, and betanaphthol may interfere with this reaction.

Fat.—Normal urine does not contain fat, but it is present in small amounts, rarely in large quantities in chronic parenchymatous nephritis, occasionally when fat occurs in excessive amounts in the blood, and after the administration of large doses of cod-liver oil. It has been observed in bone diseases in which there is a destruction of the bone-marrow, in diabetes mellitus, leukæmia, pancreatic diseases, chronic tuberculosis of the lungs, and obesity. In chyluria or galacturia the milky appearance of the urine is due to fat globules. Chylous or chyliform urine, in addition to fat, may also contain leucocytes, red blood-cells, fibrin, albumin, and occasionally leucin, tyrosin, and cholesterin.

Quinine.—The patient is to pass his water in the presence of a doctor or attendant. To 2 c.c. of urine in a test-tube are added a few drops of Tanret's solution. If the urine contains quinine it immediately assumes an opalescent appearance which is marked in proportion to the amount of quinine present. The same reaction occurs with alkaloids and albumin, but disappears in either case upon the addition of a few drops of alcohol. The test is extremely sensitive and the reaction takes place about two hours after the ingestion of quinine and lasts twenty-four hours after taking quinine and reappears during twenty-four hours, even when small quantities of quinine have been ingested, and as long as forty-eight hours with 1.50 or 2.00 gramme doses. This test is important, especially in military and institutional life, for the following reasons:

(a) To ascertain whether or not a patient in whom symptoms have not been relieved has actually taken the daily doses dispensed to him.

(b) To ascertain whether or not quinine administered in pill form or capsules has actually been freed in the intestines or dissolved.

(c) To ascertain in a case where sufficient quinine has been administered for the proper length of time and the symptoms are not relieved, whether or not they are due to malaria.

Cryoscopy of the Urine.—The determination of the freezing point of the urine permits one to measure its molecular concentration. The apparatus devised by Beckmann is generally employed in ascertaining the freezing point. The average freezing point in normal individuals, as determined by Koranyi, is -1.7° C., although wide variations are noted. Cryoscopy of the urine is rarely employed in routine clinical work, since the results have not been satisfactory.

Cammidge's Test.—A test for the detection of pancreatic disease has been suggested by Cammidge. He holds that this reaction is due to the presence in the urine of a peculiar body, probably pentose. For the details of this elaborate procedure the reader is referred to the manuals on special laboratory work. Cammidge claimed that a positive reaction occurs in all cases of active inflammation of the pancreas, and that by this means acute inflammation of the pancreas can be differentiated from intestinal obstruction, and chronic inflammation of the organ giving rise to occlusion of the

common duct can be diagnosticated from gall-stone disease. It was asserted also that "a positive reaction is indicative of altered carbohydrate metabolism due to disturbance of the internal secretion of the pancreas." The enthusiasm excited by this announcement was followed by a speedy reaction.

The results of critical studies in a series of cases at the Mayo Clinic in 1911 justify the conclusion that "if knowledge of the clinical histories and other factors of the personal equation be eliminated, the end results, judged by Cammidge's own criteria, must be considered as a means of diagnosing diseases of the pancreas, to be both valueless and misleading." Similar conclusions followed prolonged and careful studies in the surgical clinic of the German Hospital in Philadelphia and elsewhere. It has been shown that rapid disintegration of any of the body cells, and especially of the polynuclear leucocytes, may cause the reaction in the urine.

Phenolsulphonaphthalein Test for Renal Function.—See page 636.

VII.

THE EXAMINATION OF THE SPUTUM.

Systematic examination of the sputum furnishes important clinical data in a considerable group of diseases (see also pp. 467–475).

MICROSCOPICAL EXAMINATION.

Leucocytes.—The mere presence of leucocytes has no special significance, since they occur in every specimen. A sputum containing an abundance of white blood-corpuscles generally indicates a pathological disturbance of some part of the respiratory tract, as chronic bronchitis, bronchiectasis, pulmonary abscess, tuberculosis with cavity formation, or may be due to a rupture of an extrapulmonary purulent collection into the lungs. The polynuclear neutrophile leucocytes are most often found in sputum, although in a limited number of diseases, particularly bronchial asthma, eosinophiles are noted. The sputum in asthma is usually loaded with eosinophiles, some of which have the characteristic morphology and staining reaction of the hæmic eosinophiles, while others are supplied with a circular nucleus. In certain cases of bronchitis, tuberculosis, and after hæmoptysis, eosinophiles are present in the expectoration.

Epithelial Cells.—Every specimen of sputum contains epithelial cells. Pavement epithelium may be derived from the mouth, the pharynx, and the upper half of the larynx, while cylindrical cells may come from the nose, the lower part of the larynx, trachea, and bronchi. Catarrhal inflammation, especially in its early stages, generally determines the presence of large numbers of epithelial elements. Ciliated cells are occasionally found in asthma and acute bronchitis, provided the specimen be examined immediately after expectoration. Alveolar epithelial cells which occur in the sputum in almost every pulmonary disease, as well as in the "so-called" normal expectoration, are large, of an oval, round, or polygonal shape, supplied with one or several relatively small vesicular nuclei, imbedded in protoplasm which often contains albuminous granules, myelin droplets, fat globules, particles derived from hæmoglobin, or coal pigment. These cells occur in abundance in acute inflammatory pulmonary disease and tuberculosis. Myelin granules have an irregular outline, often present a concentric arrangement, and are found either intra- or extracellularly. Myelin probably consists mainly of protagon and of small amounts of lecithin and of cholesterin. These droplets dissolve in alcohol, stain light yellow with iodine, poorly with aniline dyes, and are not blackened with osmic acid. Alveolar epithelium, containing granules of altered blood pigment, is seen in the sputum of congestion of the lungs, notably in that form due to heart disease, hence the term "heart disease cells" is applied to them.

Red blood-cells occurring in small numbers are commonly observed in the sputum of many diseases of the respiratory tract and, therefore, have

no special importance, but when present in considerable or large numbers indicate a morbid lesion. Expectoration of blood (hæmoptysis) is due to a variety of causes (see page 458). Erythrocytes in the sputum, as a rule, exhibit alteration of structure, so that crenated, dehæmoglobinized, and fractured cells are common.

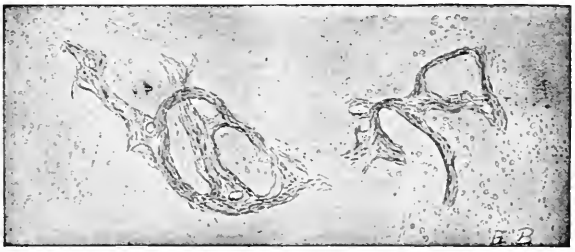


FIG. 122.—Elastic tissue from lung showing alveolar arrangement. × 50.—Emerson.

Elastic tissue, in considerable amounts, can be readily demonstrated by the following method: A thin layer of sputum, obtained by pressing it between two glass plates, is examined with the aid of a hand lens. When elastic tissue cannot be recognized by this method, the microscope should be employed; a suspected particle, which generally has a gray or yellow color, is placed upon a slide and studied by low magnification. Elastic tissue may also be demonstrated by treating the sputum with an equal quantity of a 10 per cent. solution of potassium or sodium hydroxide and boiling the mixture until it becomes homogeneous. The solution is shaken with four or five parts of water and the mixture centrifugated. The sediment is then examined microscopically. Elastic tissue is found as long slender threads, generally having a waxy appearance, and at times these fibres conform to the outline of alveoli. The presence of elastic fibres indicates disintegration of bronchial or pulmonary tissue, the latter being positively affirmed when the fibres have an alveolar arrangement. Elastic tissue is noted in bronchiectasis, pulmonary abscess, gangrene, tuberculosis, and tumors of the lungs.

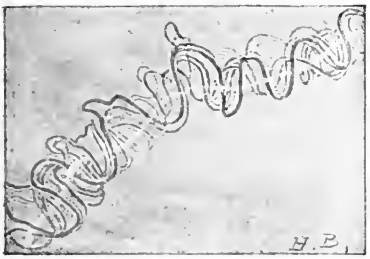


FIG. 123.—Curschmann's spiral, from the sputum of a case of asthma. × 200.—Emerson.

Curschmann's spirals are noted in the sputum in cases of bronchial asthma, occasionally in tuberculosis, croupous pneumonia, and bronchitis. Upon microscopic examination, they consist of delicate twisted threads, often wound around a central core. Many of these spirals are coated with mucus in which epithelial cells, eosinophiles, neutrophile leucocytes, and Charcot-Leyden crystals are imbedded. Curschmann's spirals consist chiefly of mucus, while the central core is held to be fibrin in some instances. Many authorities claim that these bodies are formed in the bronchioles.

Crystals.—With the exception of Charcot-Leyden crystals, very little importance can as yet be attached to the presence of crystalline bodies. Charcot-Leyden crystals are colorless and have the shape of two elongated, sharply pointed, hexagonal, pyramidal figures with bases opposed. They stain with eosin. It was formerly thought that they were the exciting factor of bronchial asthma. This view is no longer entertained, since these

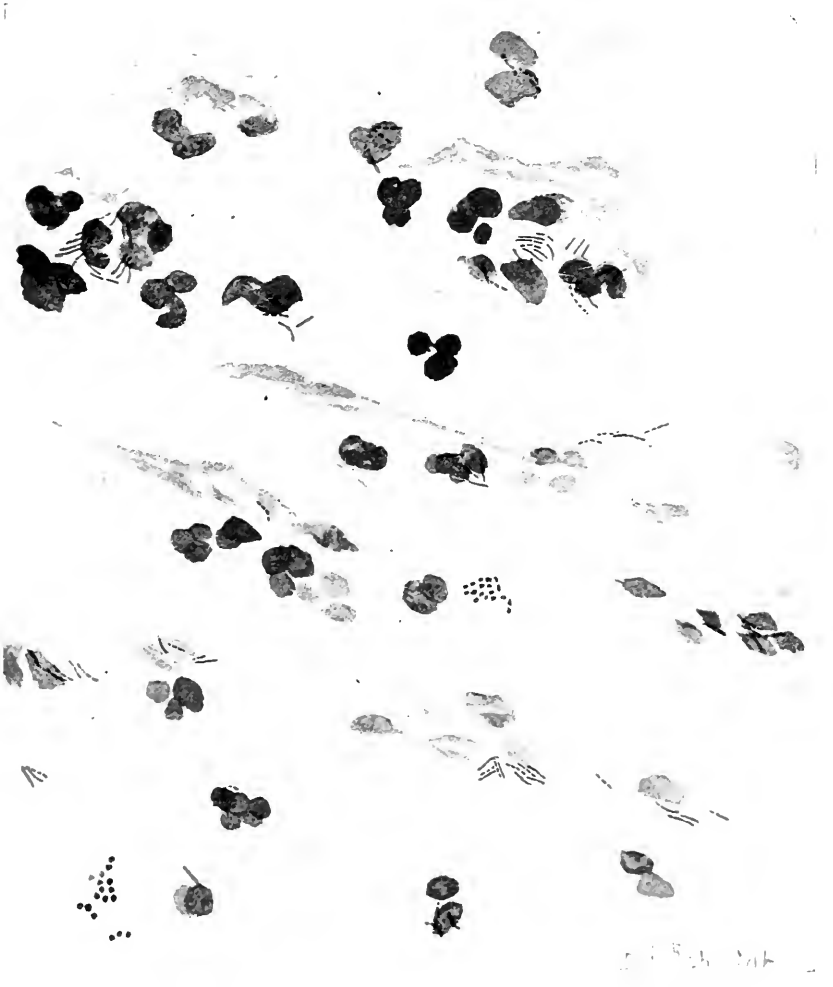
crystals are occasionally found in other diseases, such as bronchitis and tuberculosis. They are probably formed from eosinophile cells. Crystals of fatty acids are noted in the sputum of tuberculosis, gangrene, bronchiectasis, and fetid bronchitis. CHOLESTERIN PLATES, which are rarely seen in the sputum, have been found in conjunction with fatty acid crystals in abscess of the lung, and phthisis. HÆMATOIDIN CRYSTALS occur in the putrid sputum of certain lung diseases, and in empyema and hepatic abscess with a bronchial outlet, and occasionally after hæmoptysis. Leucin and tyrosin crystals are at times present in purulent sputum, while calcium oxalate and triple phosphate crystals are rare ingredients of sputum.

Animal Parasites.—THE TRICHOMONAS PULMONALIS has been reported in a few instances in the sputum in lung gangrene, tuberculosis, abscess, and putrid bronchitis, while circomonads have been recorded in pulmonary gangrene. The sputum in cases of liver abscess perforating into the lung may show the Amœba coli. TÆNIA ECHINOCOCCUS.—Hydatid disease may cause pulmonary abscess or gangrene and is sometimes responsible for copious hæmoptysis. The sputum in this condition may contain shreds of cyst membrane, daughter cysts, scolices and hooklets of the worm. DISTOMA PULMONALE.—This parasite is responsible for a form of chronic pulmonary disease, characterized by hæmoptysis, seen in Japan, China, and Korea. This fluke and its ova are found in the sputum.

Vegetable Parasites.—A large number of micro-organisms have been found in the sputum. Among these may be mentioned: the tubercle bacillus, Diplococcus pneumoniæ, staphylococci, streptococci, sarcinæ, streptothrix, actinomyces, Micrococcus catarrhalis, and the influenza, smegma, typhoid, plague, diphtheria, and Friedlander's bacillus.

In the case of tubercle bacilli, their staining reaction, outline, and size, in the absence of biological tests, generally afford sufficient evidence to establish the diagnosis of this organism. With most bacteria occurring in the sputum this is not the case, so that their identity can only be determined provisionally but not finally by their tinctorial and morphological characteristics. This tentative opinion is, however, often strengthened by the correlation of the clinical data of the underlying pathological process. Cultural studies are as a rule essential, and inoculation experiments often required for a bacteriological diagnosis. Works on bacteriology should be consulted for bacteriological investigations.

TUBERCLE BACILLUS.—The finding of tubercle bacilli in the sputum is a valuable sign in establishing the diagnosis of tuberculosis of the lungs, although the absence of these organisms in the expectoration of an individual presenting pulmonary symptoms does not necessarily negative the diagnosis. The failure to find bacilli on a number of examinations in a suspected case, particularly of a chronic nature, is strong evidence against the existence of phthisis. In acute tuberculosis, especially in the early stages, they are frequently wanting in the sputum. There is no single characteristic presented by macroscopic examination of the sputum by which its tuberculous nature can be recognized. Rosenberger holds the view based on repeated observations that tubercle bacilli are present in the fæces of persons suffering from active pulmonary tuberculosis, even in the acute miliary form. The *technic* of the examination for tubercle



Tubercle bacilli in sputum stained with carbol fuchsin and Pappenheim's reagent.

bacilli is as follows: Preferably a caseous mass or a bit of purulent or hemorrhagic sputum is placed upon a slide or cover-slip. In the absence of cheesy particles, specimens are selected from different parts of the sputum. A thin smear is made, carefully dried and fixed by rapidly passing the slide or cover-glass through a flame several times. The tubercle bacillus belongs to the group of acid-fast bacteria, which, after staining, resist to a marked degree decolorization with solutions of mineral acids.

To concentrate the bacteria in the specimen of the sputum, the method of Mülhäuser-Czaplewski will be found most serviceable. From four to eight volumes of a 0.25 per cent. solution of sodium hydrate are added to the sputum, placed in a bottle, and shaken until the fluid has a uniform mucilaginous appearance. A few drops of phenol-phthalein solution are added and the liquid is boiled. A 2 per cent. solution of acetic acid is now added drop by drop until the pink color of the liquid just disappears. The material can now be centrifugated and the sediment examined.

Antiformin.—To a portion of sputum in a centrifuge tube add an equal amount of a 10–15 per cent. solution of antiformin; allow it to stand for a few minutes and then centrifugate. Decant the supernatant antiformin solution and again centrifugate. At this point a few cubic centimetres of alcohol may be added to lower the specific gravity and aid in the precipitation of the bacilli. The sediment is then smeared on the slide and stained by one of the usual methods.

Ziehl-Neelsen Method.—The stain consists of 10 c.c. of a concentrated alcoholic solution of fuchsin, dissolved in 90 c.c. of a 5 per cent. solution of carbolic acid. The film of sputum is covered with the stain. The cover or slide is then held over a flame until the solution is brought to the boiling point; or the specimen may be stained in cold carbol fuchsin for 24 hours. After a half minute, the excess of hot stain is poured off and the specimen washed with water. The stained preparation is next placed in a 25 per cent. solution of nitric acid for several seconds until the bright red color disappears, then washed in water and dried. The specimen may be counterstained with a watery solution of Bismarck brown or methylene blue for a minute or two. The cover-glass film is mounted on a slide in balsam or cedar oil. The specimen spread and stained upon a slide, the most convenient method, may be examined without a cover-glass.

Gabbett's Method.—The sputum properly spread and fixed upon a slide or cover-glass is covered with a reagent consisting of fuchsin 1 gramme, alcohol 10 cubic centimetres, and a 5 per cent. solution of carbolic acid 100 cubic centimetres, and held over a flame until the stain boils. After draining off the carbol fuchsin from the slide, the specimen is treated for two minutes with Gabbett's reagent, composed of methylene blue, 2 parts, dissolved in 100 parts of a 25 per cent. solution of sulphuric acid; then washed with water, thoroughly dried, and examined microscopically.

Pappenheim's Method. — This method affords the means of distinguishing tubercle bacilli from other acid-fast organisms. The stain is prepared by dissolving 1 part of corallin in 100 parts of absolute alcohol. This solution is then saturated with methylene blue, after which 20 parts of glycerin are added. After staining the specimen with a heated carbol fuchsin solution in the manner previously described,

the excess of stain is drained from the slide and immediately Pappenheim's solution is placed upon it and allowed to act for a few minutes. Fresh solutions may be added several times if the spread is tinged red in any part. The slide is next washed in water, dried, and examined.

With these methods, tubercle bacilli appear as straight or slightly bent red rods, varying from 1.5 to 4 microns in length and from .1 to .2 micron in thickness. Occasionally they are tinted more deeply in certain parts, having the appearance of a streptococcus (beaded forms). Branching forms are rarely found. The older varieties of bacilli are thought to retain more intensely than the younger forms. As a rule a number of organisms can be found in preparations, many of which are frequently arranged in groups containing several or more organisms. It is most uncommon to find but a single bacillus in a specimen and, when this occurs, the possibility of contamination of the sputum from dust should be remembered. The number of germs in chronic cases often is an index to the extent of the ulceration in the lung, although, in acute cases, the degree of the tubercle involvement bears no relation to the abundance of bacilli. A lessening in the number of bacilli ofttimes is associated with a steady improvement in the patient, and a disappearance of the micro-organisms frequently points to quiescent or healed lesions.

Diplococcus Pneumoniæ.—The finding of pneumococci in the sputum, in the absence of other clinical data, is without diagnostic significance, since these organisms exist in the saliva of a considerable proportion of healthy individuals, as well as in the expectorated material in several diseases. Their presence in the sputum of a case exhibiting pulmonary symptoms often establishes an etiological diagnosis. Pneumococci are found in large numbers in the sputum of croupous pneumonia and occasionally in bronchopneumonia. This organism reacts positively to solutions of basic dyes. Stained specimens frequently show a colorless capsule about the diplococci.

VIII.

THE EXAMINATION OF TRANSUDATES, EXUDATES, AND THE CONTENTS OF CYSTS.

The results of the examination of transudates, exudates, and the contents of cysts by physical, chemical, microscopical, and bacteriological methods are diagnostic auxiliaries.

Exploratory Puncture.—An exploratory syringe, equipped with a large stout needle, is generally used for this purpose, but for some explorations the aspirator needle alone is employed, since the positive internal pressure of certain effusions expels the fluid. The operation of exploratory puncture must be performed under strict antiseptic precautions; the skin should be sterilized by thoroughly scrubbing with soap and hot sterilized water, followed by washing with hot sterilized water and then

with an antiseptic solution. When possible an antiseptic dressing should be applied for some hours prior to performing the operation. The hands of the operator should be surgically clean and the instrument should be sterile. The technic of peritoneal, pleural, pericardial, and lumbar puncture is discussed in Part IV.

The differentiation between exudates and transudates is not, as a rule, difficult, since the internist is guided by associated clinical phenomena in determining the origin of the fluid. There are, however, cases in which the character (whether it be inflammatory or non-inflammatory) of the material cannot be ascertained by the symptoms or the history of the case, and the final distinction must rest with the laboratory, although in a few instances the various methods of laboratory research fail to solve the problem.

Transudates are generally light yellow or pale yellowish-green, at times reddish, due to blood staining, milky as noted in chylous effusions, and dark yellow when deeply tinged with biliary pigment.

The composition of transudates of the peritoneal, pleural, and pericardial sacs is nearly the same. They consist of water (95–96 per cent.), solids, proteids, extractives, inorganic salts, and uric acid. Allantoin, dextrose, fructose, urobilin, and biliary pigment have also been demonstrated in transudates. Their specific gravity is generally below 1.018, in many cases as low or even below 1.010, and occasionally above 1.020. Hydræmic transudates are of lighter specific gravity than those due to stasis. The specific gravity is influenced mainly by the proteids contained in the fluid, so that exudates, which are generally richer in albuminous bodies than dropsical fluids, are as a rule of higher gravity than transudates. The estimation of the total proteids content is therefore of value in differentiating between transudates and exudates. The percentage of proteids in stasis transudates generally ranges from 1 to 3, while in hydræmic effusion it is much lower, usually not above .5. Transudates either contain no fibrin or it exists only in minute amounts. A few endothelial cells and leucocytes, at times erythrocytes and cholesterin crystals, are found. In hydroperitoneum occurring in leukæmia, Charcot-Leyden crystals, mast cells, and eosinophiles have been recorded. In the main the chlorides exist in greater concentration in transudates than in exudates, and as a rule the degree of alkalinity of dropsical fluids is about that of the blood of the individual in question, while in an exudate it is lowered.

Exudates.—The chief varieties of exudates are serous, hemorrhagic, purulent, and putrid, and between these types there are gradations and combinations. The recognition by macroscopic inspection of purulent collections is generally a simple matter, although serous exudates, which contain a large number of fine fibrin flakes and chylous fluids, are of similar appearance. The uniform turbidity of purulent effusions serves to distinguish them from serofibrinous effusions, while the presence of fine granules of fat is characteristic of chylous fluids. In many inflammatory collections a coagulum forms immediately after the fluid is withdrawn from the body. Their specific gravity is generally above 1.018, the proteid content is usually above 4 per cent. and at times as high as 6 per cent. Serum albumin and globulin in considerable amounts, traces of fibrinogen

and serosamucin are present in exudates; nucleo-albumin, albumoses, leucin, and tyrosin have also been noted.

Rivalta's Test.—The principle of this test is based on the precipitation in many exudates of a peculiar body, the character of which has not been definitely determined, although regarded by some authorities as mucin and denominated serosamucin, while others hold that it is a globulin. This test is carried out by allowing a drop of the fluid to fall into a weak acetic acid solution (two drops of glacial acetic acid in 100 c.c. of distilled water). When the drop sinks and leaves a turbidity it indicates the presence of this substance, while the failure to produce cloudiness denotes the absence of this body (serosamucin). The intensity of the cloudiness and the rapidity with which it forms are an index to the amount present. This test is of importance in differentiating exudates from transudates.

Animal parasites, bacteria, many cellular elements, lymphocytes, polynuclear cells, endothelial cells, erythrocytes, and detritus occur in exudates.

Bacteriological Examination. — Bacteria rarely exist in transudates, but their presence in exudates, which is frequent, furnishes a most useful field for diagnosis and prognosis. The fluid for bacteriological examination is collected in a sterile flask, the neck of which is then immediately plugged with sterile cotton. (For technic consult works on bacteriology.) A diagnosis of tubercle bacilli can often be made by staining methods.

Collecting Sediment.—Fluids removed by puncture often coagulate spontaneously. Since the coagulum entangles some of the cellular bodies and bacteria, the elements which remain in the fluid portion do not form an accurate basis for calculating the number or the percentages of the different varieties of cells. In order to prevent coagulation one-third or fourth volume of a 2 per cent. sodium citrate salt solution is added to the specimen. After centrifugalization or sedimentation the supernatant fluid is removed and the tube is filled with saline solution, then gently agitated and recentrifugated. Much of the albumin is removed from the fluid by this procedure, which insures better results in staining.

Inoscopy, the method introduced by Jousset, was designed to aid in the diagnosis of tuberculosis. The exudate is allowed to coagulate spontaneously, but should this not occur the addition of horse serum will bring about clotting. The coagulum which holds many of the tubercle bacilli is then removed, broken up, and digested by means of a fluid consisting of NaF 3 grammes, pepsin 1 or 2 grammes, glycerin 10 c.c., HCl 40 per cent. 15 c.c., water 1000 c.c. The resulting liquid is then centrifugated and the sediment examined in the usual manner for tubercle bacilli.

Cytological Examination. — After securing the sediment of the citrated material, or the digested coagulum, it should be properly fixed. Treating the sediment with a $\frac{1}{2}$ or 1 per cent. formaldehyde solution for several minutes is highly recommended by some workers. The sediment is spread into a thin film upon a slide or cover-glass, dried, and if not previously fixed is now subjected to such fixatives as methyl alcohol, or alcohol and ether, heat, or formalin solutions. The selection of the stain depends upon the structures desired to be demonstrated and upon the choice of the worker. Most of the Romanowsky modifications or double stains, as eosin and hæmatoxylon or methylene blue, give satisfactory results. The

principle which involves the determination of the percentages of the various types of cells is the same as for the differential counting of leucocytes. Immediate citration of fresh specimens, followed by centrifugating, probably offers the best means of studying cellular elements and bacteria.

Cytodiagnosis.—The cytological formula does not diagnose a disease, but rather suggests the acuteness or chronicity of a pathological condition, the stage and intensity of a morbid process, or the absence of inflammation. A rare exception relates to effusions which contain tumor fragments, the histology of which may be diagnostic.

The most important cellular elements entering into cytological studies are lymphocytes, polynuclear cells, endothelial cells, eosinophile cells, mast cells, erythrocytes, and tumor cells.

Endothelial Cells.—An increase of endothelial cells in a fluid is generally associated with non-inflammatory effusions of the serous cavities. Dropsical effusion due to passive congestion and hydræmic transudates shows endotheliocytosis. In the early stages of a tuberculous effusion a high percentage of endothelial elements is sometimes noted.

Lymphocytes. — An irritant of mild intensity is responsible for a lymphocyte predominance in an effusion. Such a reaction is essentially local and does not provoke a general stimulus. An irritation of low grade, especially when protracted over a long period, calls forth these cells. Lymphocytosis is the rule in tuberculosis, although a polynucleosis may precede a lymphocytic phase, or in some instances it may follow. These variations are attributed to increased virulence of bacteria and to secondary or mixed infections. A lymphocyte preponderance preceded by a polynucleosis is regarded as having a favorable prognostic significance. The development of a polynucleosis taking the place of a lymphocytosis is suggestive of a complication. In the late stages of acute inflammations or when these tend to become chronic, a high lymphocyte percentage is often noted. Lymphocytosis is noted almost constantly in effusions of tuberculous origin and sometimes in those due to syphilis, uræmia, malignant tumors, and paresis.

Polynuclear Cells.—The exudates in acute inflammation or infections of serous sacs, such as are produced by staphylococci, pneumococci, streptococci, meningococci, colon bacilli, and typhoid bacilli, contain a high percentage of polynuclear leucocytes. In the early stage of tuberculosis a polynucleosis is sometimes noted, and frequently in tuberculosis pericardial effusions. As an acute inflammation subsides polynuclear preponderance becomes less marked, and this is often followed by a rise in the number of the lymphocytes, which may outnumber the multinuclear elements.

Eosinophilic cell increase has been recorded in effusion occurring in the course of rheumatic fever, tuberculosis, nephritis, syphilis, carcinoma, and following trauma.

Mast cells have been noted occasionally in effusions, especially those of long standing.

Erythrocytes.—Contamination of the fluid with blood from the wound

made by puncture is unavoidable in many instances, but aside from this source red corpuscles in an effusion are at times the expression of malignant, renal, or tuberculous disease. They are also seen in effusion due to acute infections. They may be seen in acute leukæmia and the blood dyscrasias. The possibility of a hemorrhage, as in cerebral apoplexy with effusion into the ventricles of the brain, or a small leak of an aneurism into a serous sac, should always be borne in mind.

The recognition of some of the varieties of cells just described may not be so simple a matter. A cell having a single nucleus undergoing degeneration and fragmentation may resemble a multinuclear element. Polynuclear cells may be difficult to detect when the cell body undergoes shrinkage and becomes disintegrated.

Chylous fluids owe their turbidity to fine particles of fat. The amount of fat varies; it is often under 1 per cent., but in a case reported by Hammerfahr it reached 2.95 per cent. Other constituents of this variety of effusion are water (90 per cent. +), albumin, fibrin, globulin, cholesterin, lecithin, salts, soaps, fatty acids, and other substances. The fat is soluble in ether and gives the tests for this substance.

There are certain effusions designated chyloid or pseudochylous which closely resemble chylous fluids in their gross appearance but differ from them since the free, fine, fat particles are absent. The opalescence of these fluids probably depends on a variety of causes, while in some instances the milky appearance cannot be explained. The presence of endothelial or epithelioid cells with a fatty degenerated protoplasm is the explanation suggested by Quincke in some of these cases. Other observers hold that bacteria, globulins, lecithin, mucin, and certain proteids (other than globulin) are responsible for the turbidity which may in some instances suggest a purulent character rather than a milky appearance.

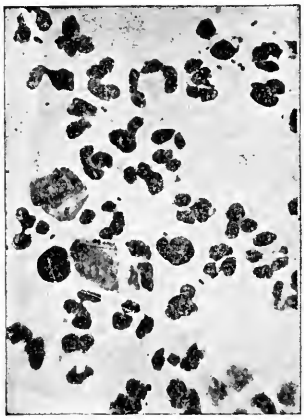


Fig. 124.—Smear of the spinal fluid of a case of epidemic cerebrospinal meningitis.—Emerson.

Chylous collections are not uncommonly noted, especially in the peritoneal cavity and pleural sacs, rarely in the pericardium. These effusions arise in a number of diseases in which pressure is exerted on the thoracic duct or the lymphatic vessels.

Cerebrospinal Fluid.—In health the cerebrospinal fluid obtained by lumbar puncture is colorless, clear, of alkaline reaction, has a low specific gravity, ranging from 1.003 to 1.007 due to the presence of from 1 to 1.5 per cent. of solids and cellular elements (endothelial cells and leucocytes), not exceeding 5 per c. mm. The amount under normal conditions has been set as varying between 5 and 10 c.c. although these figures are only approximate. The dural pressure as determined with an ordinary water manometer in the dorsal position ranges from 60 to 100 mm. in health, while in disease, as in meningitis and cerebral tumor, it may reach from 200 to 800 mm. Serious symptoms may arise on withdrawing the fluid when the pressure falls below 60 mm.

Urea, globulin, protalbumose, nucleoproteid, and a reducing substance probably similar to pyrocatechin, and sodium chloride and other inorganic salts are present. Serum albumin is said never to exist in the normal fluid. There is some doubt as to the presence of glucose; some authorities claim that it is a normal constituent (.4 to .5 per cent.), which disappears when meningitis develops (Lannois and Boulard), while others hold that it does not exist in health.

In pathological conditions the cerebrospinal fluid is often altered.

In disease the quantity varies from a few c.c. to more than 100 c.c. The amount is increased in acute hydrocephalus, in general paresis, dementia præcox, in some of the infectious diseases, in brain tumors, and in meningitis. It should be borne in mind that interference with the circulation of the cerebrospinal fluid from increased intracranial pressure, as in brain tumor, which cuts off the communication between the subarachnoid basilar spaces of the brain and those of the cord, may result in an absence or diminished amount.

The fluid is often pale yellow, cloudy, or creamy in appearance in acute meningitis, while in tuberculous meningitis, hydrocephalus, and brain tumors it is generally clear and colorless. In hemorrhage into the ventricles fluid blood may be obtained by puncture, while in icterus the fluid is yellowish. As a rule a turbid fluid points to an acute meningitis. Albumin may be present and the specific gravity is raised when acute inflammation of the meninges exists. Cholin, a substance which is derived from the destruction of nerve tissue, is present in the spinal fluid in cases of organic disease of the nervous system, notably in paresis, tabes dorsalis, syphilitic epilepsy, dementia paralytica, cerebral abscess, brain syphilis, myelitis, and spina bifida.

Bacteriological studies of the spinal fluid are most essential in diagnosis. The following are the more important bacteria which have been found by lumbar puncture: meningococcus, pneumococcus, staphylococcus, streptococcus, B. tuberculosis, B. coli communis, B. influenzæ, B. mallei, B. pyogenes fœtidus.

Trypanosomes are present in the spinal fluid in African sleeping sickness.

Cytological Examination.—The results of cytological studies of the cerebrospinal fluid fall in line with those previously mentioned. In tuberculosis a high lymphocyte count is the rule. Lymphocyte preponderance has also been noted in paresis, tabes, cerebrospinal syphilis, syringomyelia, cerebral tumors, pressure myelitis, in chronic and in later stages of cerebrospinal meningitis, in epilepsy, and in sleeping sickness. In acute meningitis, such as is determined by the meningococcus, staphylococcus, streptococcus, pneumococcus, B. typhosus, B. coli communis, a multinuclear cellular predominance exists.

Contents of Cysts.—Pancreatic Cysts.—The evidence that the fluid from an abdominal cyst has the property of digesting albumin in an alkaline medium suggests a pancreatic origin. A negative result does not rule out the possibility of pancreatic cyst, since trypsin disappears in collections of long standing.

Ovarian Cyst.—Fluid of ovarian cysts is often pale yellow, sometimes

reddish or dark brown; the specific gravity shows wide fluctuation between 1.010 to 1.038; the consistency varies from a watery fluid to dense, viscid, "jelly-like" material.

Cystic collections of low specific gravity contain little albumin (serum albumin and globulin), while those of high specific gravity have large amounts of albumin. Mucin is present in colloid cysts. Pseudomucin or metalbumin also exists in these cysts.

Ciliated cylindrical epithelial cells, squamous epithelium, erythrocytes, fat, fatty acid crystals, cholesterin plates, and hæmatoidin are also noted in the cysts.

Hydatid Cysts.—They may be recognized by the presence of cyst membrane, scolices, and hooklets. The fluid of these cysts is almost colorless, of very low specific gravity, contains little or no albumin, shows a considerable amount of sodium chloride, has a neutral or faintly acid reaction, and traces of sugar and succinic acid may be present. Granular and fatty detritus, calcareous fragments, hæmatoidin, cholesterin crystals, and granular cells are frequently found. In the event of suppuration leucocytes appear in the fluid.

Hydronephrosis.—The fluid of hydronephrosis does not always present features which are diagnostic. This applies especially to chronic hydronephrosis with complete occlusion of the ureter. In acute cases or those associated with partial occlusion of the ureter so that the kidney still functionates, the presence of a high urea content and uric acid, and especially when renal tube-casts and cells are found, renders the diagnosis a comparatively simple matter.

IX.

THE EXAMINATION OF THE NERVOUS SYSTEM.

PRELIMINARY CONSIDERATIONS.

The diagnosis of disease of the nervous system demands an accurate knowledge of the anatomy and physiology of the structures involved and of the pathological processes to which they are liable. It is essential to determine not only the location but also when possible the nature of the lesion.

The nervous system, by which the organism is brought into relation with its environment and by which its functions are made manifest and controlled, is essentially composed of morphological units having a similar structure—the neurons—and held together and supported by a special tissue—the neuroglia.

The Neuron.—Each neuron consists of (a) a nucleated protoplasmic mass—the cell-body—which presides over the nutrition of the neuron and is the seat of origin of nervous impulse, and (b) processes which form outgrowths from the cell-body and constitute the elements along which impulses are conveyed. These processes are of two kinds, (a) branched

protoplasmic outgrowths, *dendrites*, which may be multiple and form arborescent interlacing ramifications with similar processes from other neurons, and (b) the single elongated process, *axon*, commonly prolonged to form the axis-cylinder process.

The dendrites, uneven in contour and relatively thick as they arise from the cell-body, rapidly become more slender in consequence of their repeated branching until they terminate in delicate end branches with terminal bead-like thickenings.

The axons are slender thread-like extensions of uniform diameter and variable length, sometimes reaching only to adjacent neurons, sometimes extending to distant neurons within the cerebrospinal axis, as from the cerebral cortex to the lower part of the spinal cord, sometimes beyond as from the lower part of the cord to the muscles of the foot. The axons, like the dendrites, finally terminate in end arborizations—*telodendria*. Most of them, shortly after leaving the cell-body, give off processes termed collaterals which after a variable course terminate in end arborizations which interlace with the processes of other and sometimes distant neurons. Less frequently short axons arise which are not continued as axis-cylinders but at once terminate in complex branching end brushes within the substance of the gray matter.

Histologists are not of accord as to whether the relation between the neurons is that of continuity or simple contiguity. The weight of opinion is at present in favor of the view that the neurons are separate and distinct morphological units, their processes interlaced to form paths of conduction but probably never actually continuous in the anatomical sense. The axis-cylinders, usually supplied with a medullary sheath, are described as *nerve-fibres*. Collected into bundles they form the *nerve-trunks* which ramify to the various muscles and other organs.

Divisions of the Nervous System.—*Central Portion.*—In vertebrates there is an axial accumulation of the cell-bodies in the cerebrospinal axis from and to which the processes pass. This includes the brain and spinal cord and contains the principal axial collections of neurons.

Peripheral Portion.—This division embraces the nerve-cells of the sensory ganglia and is chiefly made up of the nerve-fibres which pass to and from the end organs.

Sympathetic Nervous System.—This division is intimately correlated with the peripheral nervous system, but possesses a certain degree of physiological independence and supplies the unstriped muscular and the glandular tissues of the body and the muscle of the heart.

Nerve Terminations.—The terminal end arborizations of the peripheral nerves constitute the mechanism by which the various structures of the body are combined in consistent and harmonious relation with the nervous system. Certain of these terminations transmit impulses which give rise to muscular contraction; others originate impulses which cause various sensations of pain, temperature, pressure, or the special senses. The nerve terminations may therefore be divided according to their function into *motor* and *sensory*.

Motor Nerve Endings.—These include three groups: (a) The terminal arborization of the axons of neurons in the motor nuclei of the spinal cord

and brain stem that pass to voluntary muscle; (b) those of sympathetic neurons that pass to involuntary muscle; (c) the muscle of the heart.

Sensory Nerve Endings.—These are the peripheral terminal arborizations of the neurons, the cell-bodies of which are in the spinal and other sensory ganglia. They therefore constitute the point of departure of the paths which conduct sensory stimuli to the central nervous system.

The function of the neuron is to conduct nervous impulses. In its simplest form the nervous system consists of (a) the *sensory neuron,* which receives the external stimulus acting upon the integument and other sensory surfaces and by means of its process conducts it from the periphery to the cell-body which commonly lies in the cerebrospinal axis. Such a process constitutes functionally a centripetal or afferent fibre. The stimulus thus received is transmitted from the cell-body of the sensory neuron by means of its dendrites which interlace with those of the associated cell-body of (b) a *motor neuron* to the latter, in which a responsive impulse originates and is conveyed along its axis-cylinder process—nerve-fibre—to the muscle-cell and causes contraction. The latter process is therefore known as a centrifugal or efferent fibre. This elementary conception of the relation and functions of the sensory and motor neurons is greatly modified by the fact that the centripetal impulses are conveyed to the cell-bodies of other neurons not only in the immediate neighborhood but also at different and even distant levels. Neurons of the same function are usually grouped together, aggregations of cell-bodies forming nuclei, and collections of the fibres forming bundles, tracts, or systems. The former are situated in the gray matter of the brain and spinal cord, the latter run in the white substance of the brain and spinal cord and in the peripheral nerves. By this means the various parts of the central nervous system are connected with each other and with the muscles and viscera.

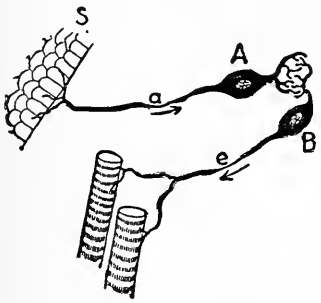


FIG. 125.—Diagram showing fundamental units of nervous system. *A*, sensory neuron, conducting afferent impulses by its process (*a*) from periphery (*S*); *B*, motor neuron sending efferent impulses by its process (*e*) to muscle.—Piersol.

Many of the tracts are highly complicated and obscure both as to their course and formation. Others are simpler and, as the result of studies of the degenerations caused by injury or disease, have been traced in their course through the cerebrospinal axis. Chief among the latter group is the pyramidal tract which transmits motor impulses from the cortex to the periphery.

The Motor System.—A muscular movement depends upon the combined functional activity of many associated neurons. It follows that the movements of the various parts of the body are represented in the central nervous system by localized aggregations of correlated neurons, or centres. Muscular movements are not only localized in the motor areas of the cerebral cortex but they are also localized in the different levels of the ventral horns of the spinal cord and the motor nuclei of the cerebral nerves. Voluntary motor impulses originating in the cortex of the brain

pass through at least two neurons before they reach the muscles. For this reason the motor tract is divided into an upper and a lower segment.

THE UPPER MOTOR SEGMENT.—Clinical researches (Hughlings Jackson), experimental studies (Hitzig, Ferrier, Horsley and others), and the studies of tract myelination at progressive periods in the development of the cerebrum by Flechsig, have thrown much light upon the functions of many of the cortical regions of the brain and the sensory and motor tracts. The cell-bodies of the upper motor neurons are arranged in functionally allied groups in the cerebral cortex over the ascending frontal convolution and extending deeply into the fissure of Rolando. In this region the movements of the body are definitely represented. It has been demonstrated that motor impulses are excited by stimulation over these areas in a definite order from above downward, as follows: leg, trunk, arm, neck, face; the areas for the leg, trunk, and arm covering the upper half, including the Rolandic surface of the convolution, and those for the head and face, together with those for the jaws, lips, tongue, and larynx, the lower half, likewise the surface extending into the fissure. The centre for motor speech lies in the left third frontal, Broca's, convolution.

The axis-cylinder processes of the upper motor neurons pass from the gray matter of the motor cortex into the white matter of the brain and form part of the extensive converging tract known as the corona radiata. Collected into a compact bundle—the pyramidal tract—they pass between the basal ganglia in the internal capsule occupying the knee and the anterior two-thirds of the posterior limb. The movements of the opposite side of the body are represented at this level from before backward in the following order: eyes, head, tongue, mouth; shoulder, elbow, wrist, fingers, thumb; trunk; hip, ankle, knee, toes.

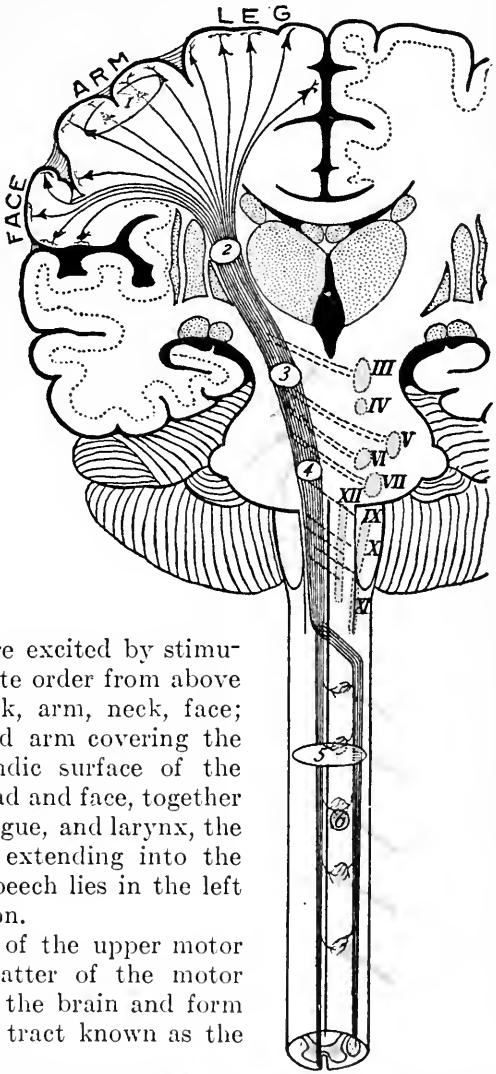


Fig. 126.—Diagram of motor path from right cortex. Upper segment black; lower red. A destructive lesion at 1 causes upper segment paralysis of the arm of the opposite side; at 2 upper segment paralysis of the opposite side—hemiplegia; at 3 upper segment paralysis of the face, arm, and leg of the opposite side and lower segment paralysis of the eye muscles of the same side—crossed paralysis; at 4 upper segment paralysis of arm and leg of the opposite side and lower segment paralysis of the face and external rectus of the same side—crossed paralysis; at 5 upper segment paralysis of all muscles below lesion and lower segment paralysis of muscles represented at level of lesion—spinal paraplegia; at 6 lower segment paralysis of muscles represented at level of lesion—anterior poliomyelitis.—VanGehuchten modified.

Emerging from the internal capsule the fibres of the pyramidal (corticospinal) tract pass into the crus. At this point some of them leave the

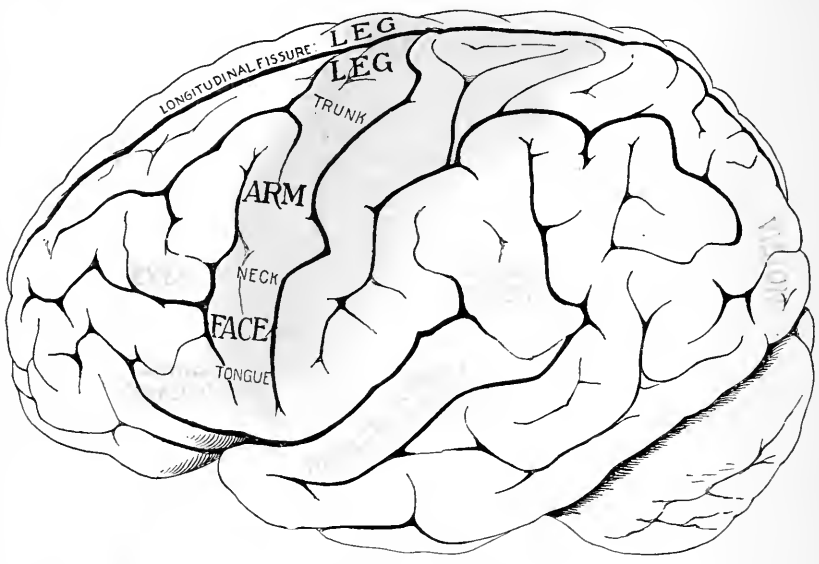


FIG. 127.—Diagram of cortical centres.

tract and crossing the middle line end in arborizations among the ganglion cells in the nucleus of the third nerve upon the opposite side, and at successive levels fibres are given off which terminate in the nuclei of all the motor cerebral nerves of the opposite side, while a limited number of fibres are distributed to the corresponding nuclei of the same side. From the crus the pyramidal tract enters the pons and passes to the medulla oblongata forming its anterior area—the pyramid. At the lower limit of the medulla, after the fibres to the nuclei of the cerebral nerves have been given off, five to seven coarse strands pass obliquely across the anterior median fissure, interlacing with similar strands from the opposite side and thus constituting the decussation of the pyramids. In consequence of this arrangement the greater number of the fibres of the important motor paths pass to the opposite sides to reach the lateral columns of the cord in which they descend as the lateral or crossed pyramidal tracts. The fibres that remain upon the same side as the pyramid from which they emerge

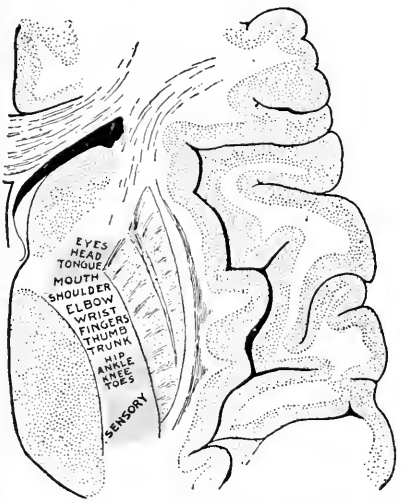


FIG. 128.—Diagram of internal capsule showing motor and sensory paths.

are collected in its lateral portion and descend in the ventral columns as the direct pyramidal tracts or Türck's columns. At every level of the spinal cord axis-cylinder processes emerge from the crossed pyramidal tract to enter the ventral horns and end in arborizations about the cell-bodies of the lower motor neurons. In consequence of this arrangement the tract diminishes in size as it descends in the cord. In a somewhat similar manner the fibres of the direct pyramidal tract cross at different levels in the ventral white commissure and end in arborizations about cell-bodies in the ventral horns on the opposite side. The direct pyramidal tract also diminishes in size as it descends and commonly ends about the middle of the thoracic portion of the cord.

Motor impulses originating in the right cerebral cortex cause muscular contractions upon the left side of the body, while those starting from the left side of the brain cause contraction of the muscles upon the right side of the body. As a rule, to which there are few exceptions, the motor paths are crossed chiefly at the decussation of the pyramids and to a less extent at different levels of the cord by fibres given off from the direct pyramidal tracts to the cell-bodies of the opposite side. This crossing in either case is in the upper motor segment.

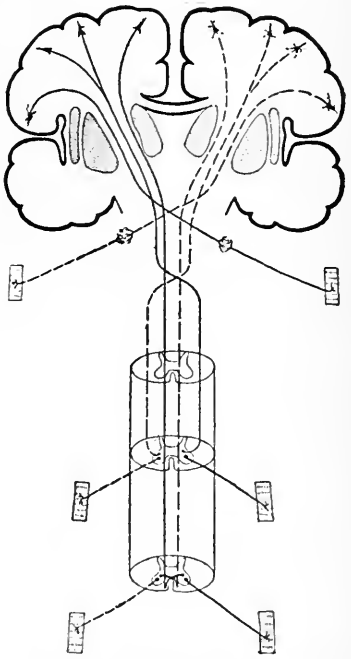


Fig. 129.—Diagram of motor path, showing the crossing of the path in the upper segment.

The Lower Motor Segment.—The cell-bodies and processes of the neurons of the lower motor segment lie in the nuclei of the cerebral motor nerves and in the various levels of the ventral horns of the spinal cord. The axis-cylinder processes of the neurons of this segment leave the spinal cord in the ventral roots and pass in the peripheral nerves to the muscles of the body, in which they end in brush-like arborizations in the motor end plates. These neurons, in contradistinction from the neurons of the upper motor segment, which are crossed, are direct, that is, the cell-bodies, their protoplasmic processes, and the muscles to which their axis-cylinders are distributed are upon the same side of the body.

The Segments of the Spinal Cord.—The spinal nerves are connected with the lateral surfaces of the cord by fan-shaped bundles of anterior and posterior roots which are collected into compact strands as they are assembled to form a common trunk. That portion of the cord to which the root fibres of a spinal nerve are attached constitutes its cord segment, the limits of which correspond to the interval which separates the extreme fibres of the nerve and those of the adjacent nerves. The spinal cord is thus seen to consist of a series of segments, each of which gives origin to the anterior or motor and receives the posterior or sensory root fibres of one pair of spinal nerves. These nerves, commonly numbering thirty-one

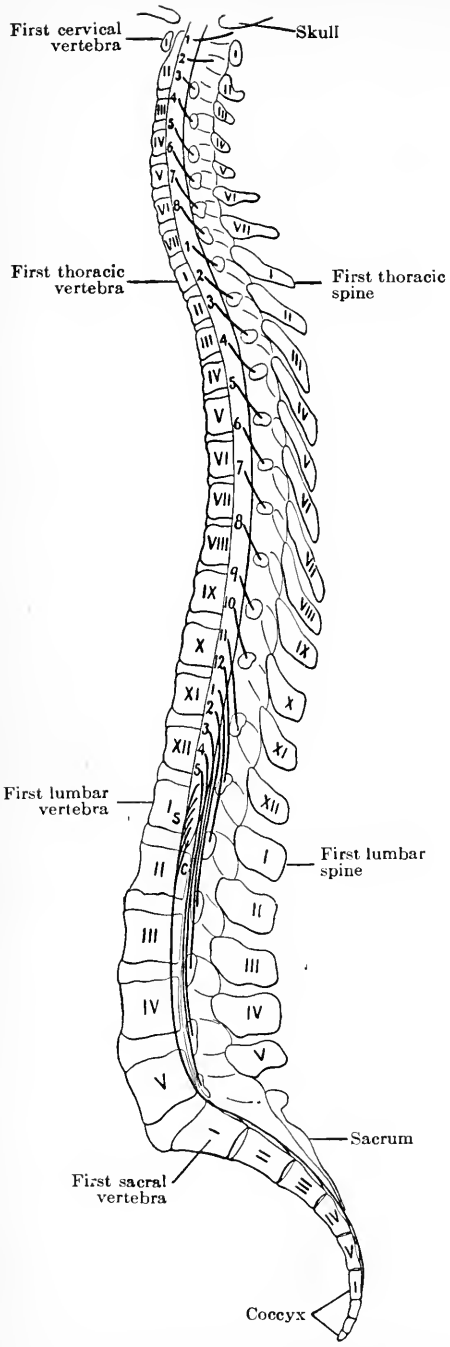


FIG. 130.—Diagram showing relations of bodies and spines of vertebræ to levels at which spinal nerves escape from vertebral canal.—Piersol.

pairs, are eight cervical, twelve thoracic, five lumbar, five sacral, and one coccygeal. In the cervical region all the nerve-roots but the eighth emerge above the vertebra, while throughout the thoracic, lumbar, and sacral regions the roots for each segment of the cord leave the spinal canal below the vertebra of corresponding number. Owing to the fact that the vertebral column increases in length to a greater extent than the cord, there is a progressive disparity from above downwards between the cord segments and their respective vertebræ. In point of fact the segment corresponds to the nerve which is connected with it, and not to the level of the vertebra opposite to it. The position of a lesion involving a particular spinal segment is therefore, except in the upper cervical region, some distance above the vertebra of corresponding number. Ziehen has formulated the following rule to determine the levels of origin of the cervical and thoracic nerve-roots: For the cervical nerves subtract one from the number of the nerve, and the remainder will indicate the corresponding spinous process; for the upper thoracic nerves (I–V) subtract two; for the lower thoracic nerves (V–XII) subtract three. Axis-cylinder processes from more than one segment of the cord may enter into the formation of a peripheral nerve and the greater number of the long striped muscles are supplied with nerve-fibres from more than one segment.

The cutaneous distribution of the peripheral nerves has been accurately worked out and

is of diagnostic value in lesions of the main trunks and their ramifications. The segmental areas which correspond to the dorsal roots, though less definitely determined, are sufficiently known to be of great service in the segmental localization of lesions of the dorsal roots and the cord. These skin-fields or dermatomes have been mapped out as

THE LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD.

Based upon the studies of Starr, Edinger, Wuhmann, and others.

Segment.	Muscles.	Reflex.
I, II, and III Cervical	Splenius capitis, trapezius, hyoid muscles, diaphragm (C. III-V), sternomastoid, levator scapulæ (C. III-V)	Diaphragmatic.
IV Cervical..	Trapezius, scaleni (C. IV-T. I), rhomboid, diaphragm, teres minor, levator scapulæ, supraspinatus	Dilatation of the pupil (C. IV-VII).
V Cervical ...	Diaphragm, rhomboid, biceps, supinator brevis (C. V-VII), teres minor, subscapularis, brachialis anticus, pectoralis (clavicular part), supra- and infraspinatus (C. V-VI), deltoid, supinator longus (C. V-VII), serratus magnus	Scapular (C. V-T. I), supinator longus (C. V), and biceps (C. V-VI).
VI Cervical ..	Teres minor and major, biceps, supinator brevis, coraco-brachialis, extensors of wrist (C. VI-VIII), infraspinatus, b achialis anticus, pectoralis (clavicular part), pronator teres, deltoid, supinator longus, serratus magnus (C. V-VIII), triceps (outer and long heads)	Triceps and posterior wrist (C. VI-VIII).
VII Cervical..	Teres major, pectoralis major (costal part), pronators of wrist, flexors of wrist, subscapularis, pectoralis minor, triceps, latissimus dorsi (C. VI-VIII), deltoid (posterior part), serratus magnus, extensors of wrist and fingers	Scapulohumeral and anterior wrist (C. VII-VIII).
VIII Cervical	Pectoralis major (costal part), latissimus, pronator quadratus, radial lumbricales and interossei, flexors of wrist and fingers	Palmar (C. VII-T. I).
I Thoracic ...	Lumbricales and interossei, thenar and hypothenar eminences (C. VII-T. I)	
II to XII Thoracic	Muscles of back and abdomen, rectus abdominis (T. V-T. XII). transversalis (T. VII-L. I), erectores spinæ (T. I-L. V), external oblique (T. V-XII), intercostals (T. I-T. XII), internal oblique (T. VII-L. I)	Epigastric (T. IV-VII), abdominal (T. VII-XII).
I Lumbar	Lower part of external and internal oblique and transversalis, psoas major and minor (?), quadratus lumborum (L. I-II), cremaster	Cremasteric (L. I-III).
II Lumbar ...	Psoas major and minor, sartorius (lower part), iliacus, flexors of knee (Remak), pectineus, adductor longus and brevis	
III Lumbar ..	Sartorius (lower part), inner rotators of thigh, adductors of thigh, abductors of thigh, quadriceps femoris (L. II-L. IV)	Patellar tendon (L. II-IV).
IV Lumbar ..	Flexors of knee (Ferrier), abductors of thigh, quadriceps femoris, extensors of ankle (tibialis anticus), adductors of thigh, glutei (medius and minor)	Gluteal (L. IV-V).
V Lumbar ...	Flexors of knee (hamstring muscles) (L. IV-S. II), flexors of ankle (gastrocnemius and soleus) (L. IV-S. II), outward rotators of thigh, extensors of toes (L. IV-S. I), glutei, peronæi	
I to II Sacral.	Flexors of ankle (L. V-S. II), intrinsic muscles of foot, long flexor of toes (L. V-S. II), peronæi	Foot reflex (S. I-II), plantar (S. II-III).
III to V Sacral	Perineal muscles, levator and sphincter ani (S. I-III).....	Vesical (L. IV-V) and anal (S. I-III).

the result of observations by Henry Head in the distribution of the cutaneous lesions of herpes zoster and the areas of referred pain and tenderness corresponding to certain visceral lesions, and in cases of gross lesions of the cord by Starr, Kocher and others; as the result of studies of anæsthesia under similar conditions; and of morphological investigations, anatomical dissections, and experimental physiological researches. The skin areas upon the trunk form irregularly parallel zones, somewhat horizontal in the erect posture, and even more irregularly distributed elongated tracts

upon the extremities. The technic consists in the use of a blunt instrument, as the head of an ordinary toilet pin, in determining the presence of areas of abnormal sensation and defining their boundaries.

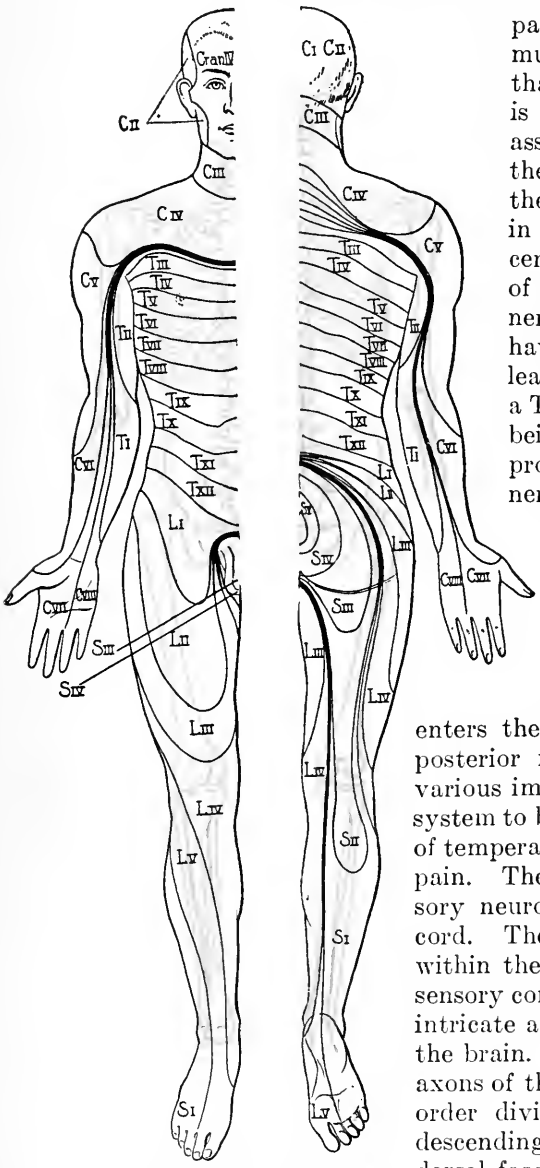


FIG. 131.—Anterior and posterior segmental skin-fields.

The Sensory System.—The path for sensory conduction is much more complicated than that for motor conduction and is composed of three or more associated neurons, one above the other. The cell-bodies of the lowest neurons are situated in the ganglia of the sensory cerebral nerves and the ganglia of the dorsal roots of the spinal nerves. The latter ganglia cells have a single process which, after leaving the cell-body, undergoes a T-shaped division, one portion being the peripherally directed process or dendrite (sensory nerve) which conducts impulses from the integument, mucous membranes, muscles, tendons, and joints of parts of the body with which it may be related; the other the axon or axis-cylinder process which enters the spinal cord by way of the posterior root fibre and conveys the various impulses to the central nervous system to be transformed into sensations of temperature, touch, muscle-sense, and pain. The larger number of the sensory neurons lie outside of the spinal cord. The portions of those neurons within the cord constitute the paths of sensory conduction, which become more intricate as the various tracts approach the brain. Upon entering the cord the axons of the sensory neurons of the first order divide into an ascending and a descending branch which run in the dorsal fasciculi. The short descending branch, after giving off a number of collaterals, terminates in the gray matter of the cord. The ascending branch is of variable length. It may soon terminate in the gray matter or may reach to the nuclei of the medulla. The lower sensory neuron does not cross the middle line. The cell-bodies about which the axis-cylinders of the neurons

of the first order and their collaterals end are the sensory neurons of the second order. The axis-cylinder processes of many of those cells cross to the opposite side of the cord and run in the ventrolateral ascending column of Gowers and the ground bundles. The lemniscus is probably the principal sensory tract in the medulla, pons, and cerebral peduncles. The fibres are not, however, continued directly to the cerebral cortex but terminate about cells in the ventrolateral portion of the optic thalamus, from which point the path of sensory conduction is continued by a higher order of neurons, the processes of which terminate in the postcentral and parietal convolutions of the cortex. Other but less direct sensory paths lie in series of neurons in the gray matter of the cord and in the direct cerebellar tract and the tract of Gowers, and pass onward through the cerebellum. Some of the axis-cylinder processes of the sensory neurons of the first order and their collaterals terminate in arborizations about the cell-bodies of the lower motor neurons and thus complete the path for reflexes.

It is probable that the conduction paths for cutaneous sensory impulses reach the opposite side soon after entering the cord, and that the paths for muscular sense lie upon the same side of the cord in the tracts of Goll, crossing by way of the axons of the second order in the medulla.

Fibre Tracts of the White Matter of the Cord.—Of these there are three sets: (1) Those which enter the cord from the periphery, viscera, and other parts of the body; (2) those which enter it from the brain; (3) those which have their origin in the cell-bodies of the neurons which lie within the cord. The fibres which arise from the same group of nerve-cells or nucleus have the same function and a similar destination and proceed together in the same course, thus constituting a tract, column, or fasciculus. Some of these fibres are the pathways for the transmission of impulses from lower to higher levels, and the strands which these form constitute ascending tracts, while others which convey impulses from above downward enter into the formation of descending tracts. These tracts are not sharply defined, nor do their boundaries and areas, since they are subject to increase and diminution by the continual accession or departure of nerve-fibres, remain the same at different levels of the cord. In fact the borders of those tracts often overlap. Their anatomical differentiation has been accomplished partly by the study of degenerative processes caused by experimental methods—Wallerian degeneration—and partly by researches in myelination at progressive periods of development—embryological method. Pathologically they are differentiated in a corresponding manner by the degenerations which follow traumatism of the cord and the definite and constant reaction of certain tracts to pathogenic influences as in tabes and other diseases.

Relation of Diseases of the Cord to Lesions of the Fibre Tracts.—In tabes and Friedreich's ataxia the posterior columns are principally involved; in combined sclerosis the posterior columns and lateral pyramidal tracts; in lateral sclerosis the lateral pyramidal tracts; in amyotrophic lateral sclerosis the lateral pyramidal tracts and the anterior horns, and in anterior poliomyelitis and progressive muscular atrophy the anterior horns.

Sensory Areas of the Cerebral Cortex.—The cortical representation of sensory stimuli is less definite than that of motion. It lies posterior to the fissure of Rolando and is extensively distributed over the post-central and parietal convolutions.

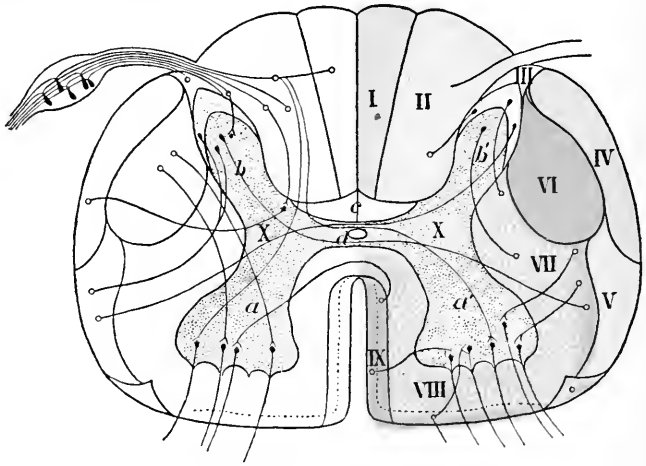


FIG. 132.—Diagram of spinal cord, showing the relation of the principal tracts.

I. Goll's or postero-internal column—fasciculus gracilus. *Termination.*—Fibres end around neurons of gray matter of cord or in nuclei of medulla. *Function.*—Sensory impulses from muscles, tendons and joints of same side. Degeneration followed by ataxia and loss of muscle sense.

II. Burdach's or posterolateral column—fasciculus cuneatus. *Termination.*—Nucleus cuneatus in the medulla; Clark's column. Collaterals to neurons of posterior horn. The root fibres passing to Clark's column traverse the middle and median part of this tract. *Function.*—Tactile impulses from opposite side. Various afferent impressions of muscle sense, heat, cold and pain. Degeneration causes pain, anæsthesia, ataxia, and loss of reflexes.

III. Lissauer's tract or marginal zone. This fasciculus is situated immediately dorsal to the inner side of the posterior horn. Composed of some of the more external root fibres which do not enter Burdach's column. Fibres of small size and short course. They penetrate the substantia Rolandi and end in arborizations about its cells and those of the caput cornu.

IV. Direct cerebellar tract—fasciculus cerebellospinalis. *Termination.*—Ascending path of the second order conveying impulses from Clark's cells to the cerebellum. *Function.*—Impulses from viscera, which probably influence maintenance of equilibrium.

V. Gowers's tract—fasciculus anterolateralis superficialis. *Termination.*—Sensory pathway of second order connecting cord with cerebellum and probably with cerebrum. Fibres are chiefly axons of neurons in the posterior horn, partly upon the same and partly upon the opposite side. Boundaries not well defined. *Function.*—The conveyance of sensory impulses—tactile pain and temperature—from opposite side by way of the anterior commissure.

VI. Lateral or crossed pyramidal tract—fasciculus cerebrospinalis lateralis. *Termination.*—Fibres are axons of cortical motor neurons. They extend from superficial gray matter of cerebrum to various levels of cord, undergoing decussation at lower part of medulla. *Function.*—Conveyance of motor impulses of brain.

VII. Lateral ground bundle—fasciculus lateralis proprius. *Terminations.*—Composition very complex. Long descending paths; one long ascending strand and many short strands both ascending and descending. *Functions.*—Both motor and sensory. Connects, by means of its intersegmental association fibres, different levels of the cord and forms a direct sensory link between cord and higher centres—medulla and cerebrum.

VIII. Anterior ground bundle—fasciculus anterior proprius. Constitutes with lateral ground bundle, with which it is continuous, a single anterolateral tract or fundamental column. Its composition and functions are the same as those of the lateral ground bundle.

IX. Anterior or direct pyramidal tract—fasciculus cerebrospinalis anterior. *Termination.*—Composed of pyramidal fibres which do not undergo decussation in medulla oblongata. Made up of 15 to 20 per cent. of pyramidal fibres. Almost all fibres cross in anterior white commissure at successive levels to terminate in arborizations about root cells of anterior horn of opposite side. *Function.*—Motor tract from cerebral cortex.

X. Gray matter of the cord. *a*, *a'*, anterior horns; emergences of anterior motor root fibres; *b*, *b'*, posterior horns; entrance of posterior root fibres; *c*, posterior commissure; *d*, anterior commissure. *Function.*—Anterior horns motor; posterior sensory. Cells of anterior horns trophic; those in angle of posterior commissure probably influence automatic movements while those near by are trophic vasomotor, and secretory.

Of the foregoing, I, II, and III comprise the fibre tracts of the posterior column; IV, V, VI, and VII the fibre tracts of the lateral column, and VIII and IX the fibre tracts of the anterior column of the cord.

The Cortical Areas for the Special Senses.—The individual sensory paths terminate in circumscribed regions which are as a rule widely removed from one another. As mapped out by myelination these areas correspond to regions of the cortex which pathological lesions have shown to be related

to the various special forms of sensation. According to Flechsig olfactory fibres end mainly in the uncinate gyrus; visual fibres have been traced to the occipital lobe in the region of the calcarine fissure, while auditory fibres run to the temporal lobe.

It is in accordance with these observations that the cuneus and calcarine fissure together constitute the primary or lower cortical visual centre in which are represented the opposite visual half fields, while the outer surface of the occipital lobe contains centres for higher visual processes in which the vision of the eye of the opposite side is represented. Mind blindness results from a destructive lesion of the lateral lobe in the left hemisphere if both occipital lobes are involved. A lesion of the cuneocalcarine cortex results in lateral homonymous hemianopsia. The centre for memory of the meaning of printed words, letters, figures, and objects seen is probably in the left angular gyrus. A destructive lesion in this area is attended by inability to read or comprehend written language although ordinary vision is not impaired. This area is known as the visual speech centre. The auditory centre is in the upper temporal convolution and transverse temporal gyri and it is in this region upon the left side that the memories of the meaning of heard words and sounds are stored. A special centre for musical memories lies anterior to the auditory centre. It is probable that the centre of each side is connected with both auditory nerves. The olfactory centre probably comprises a portion of the base of the frontal lobe and the uncinate gyrus. The gustatory centre has been thought to be in the anterior portion of the gyrus fornicatus near the centre for smell. Our knowledge in regard to these two centres is not definite.

The centres for the "higher psychical functions" are generally assumed to lie in the prefrontal lobes, particularly upon the left side. Extensive unilateral lesions of the anterior portion of the frontal lobe may be present without causing marked symptoms of any kind. Atrophy of this portion of the brain is often marked in various forms of dementia.

Symptoms due to derangements of the motor tracts constitute the most important group of localizing phenomena. They are objective on the one hand and are upon the other caused by lesions of conduction paths that are comparatively well understood. Lesions involving the motor path are irritative or destructive. The greater number of the lesions of the motor cortex are at the same time destructive and irritative. They destroy the nerve-cells and their processes in a particular centre and by their presence and advance stimulate those of adjacent centres into morbid or disordered activity. The clinical manifestation of a destructive lesion of a motor centre is loss of function—paralysis; that of an irritative lesion abnormal muscular contraction. Important differences in the paralysis or abnormal contraction are dependent upon the position of the lesion as regards the motor segments. These differences are due first to anatomical relations and second to secondary degenerations.

The cortical motor centres are more or less widely separated from one another, and a circumscribed destructive lesion of the motor area may therefore give rise to a limited paralysis involving a limb or a group of muscles in a limb—cerebral monoplegia. As the axis-cylinder processes

converge to form the pyramidal tract in the internal capsule, a lesion of limited extent causes paralysis of most of the muscles upon the oppósite side of the body—hemiplegia. A lesion in the pyramidal tract as it descends, giving off fibres to the motor nuclei at various levels, causes paralysis of the muscles having their spinal centres below the seat of the lesion. It follows from the decussation of the pyramids that when the lesion is above the crossing the paralysis is upon the opposite side of the body, and when it is below it, upon the same side.

The cell-body and particularly its nucleus maintain the nutrition of all parts of the neuron. If the cell-body be destroyed its processes undergo degeneration, or if any process be separated from its cell-body it likewise undergoes degenerative changes throughout its whole extent—secondary degeneration. Degeneration of the axons of the upper motor segment ceases, however, at the lower motor segment. The muscles are paralyzed but do not undergo degenerative atrophy; they are spastic; their reflexes are exaggerated and they do not show qualitative changes in their electrical reactions.

In complete transverse lesion of the cord—complete spinal paraplegia—the muscles upon both sides are paralyzed below the lesion, but they are flaccid; the deep reflexes are abolished; the muscles undergo rapid atrophy with loss of faradic excitability.

Irritative lesions of the upper motor segment involving the motor cortex give rise to the convulsive phenomena known as cortical or Jacksonian epilepsy.

Destructive lesions of the lower motor segment cause degeneration alike of the axis-cylinder processes in the peripheral nerves and of the muscle-fibres with which they are connected. The anatomical distribution of the cell-bodies of the segment gives rise to special peculiarities in the distribution of the paralysis which are strongly in contrast to that resulting from lesions of the upper motor segment and which have important bearings upon the localization of the lesion. These cell-bodies are collected in groups or nuclei from the peduncles of the brain throughout the entire extent of the spinal cord and send axis-cylinder processes to all the muscles of the body. Certain groups of the neurons which make up the lower segment are therefore widely separated, and a circumscribed lesion may result in paralysis of a limited number of muscles or a group of muscles instead of one-half of the body as in upper segment paralysis—hemiplegia. A lesion causing lower segment paralysis may be situated either in the cord or in the peripheral nerve. If in the cord or its nerve-roots the paralyzed muscles are not supplied by a single nerve but are represented in adjacent cord segments and the accompanying sensory derangements involve the skin fields related to those segments; if on the contrary the lesion is in the nerve, the paralyzed muscles and the anæsthetic area are those supplied by that particular nerve and its branches. The neurons of the lower motor segment maintain not only the nutrition of their axis-cylinder processes which make up the peripheral nerves but also that of the muscle-fibres in which their processes terminate. The degeneration which results from injury of the cell-bodies or their processes involves the muscles to which they are distributed. In lower motor segment

paralysis the affected muscles are the seat of degenerative atrophy, manifest in diminished tension, abolition of their reflexes and reaction of degeneration—flaccid paralysis.

Irritative lesions of the lower motor segment cause fibrillary contractions which may be due to stimulation either of the cell-bodies or of their axis-cylinder process in the peripheral nerves; or they may give rise to spasmodic contractions when the lesion affects the motor nerve-roots as they emerge from the cord.

Symptoms due to derangements of sensory paths are of far less localizing value than motor symptoms. This is partly due to the greater complexity of the sensory tracts, partly to less exact knowledge concerning them. If sensory symptoms are limited to the distribution of a peripheral nerve it is evident that the lesion is in the nerve-trunk or its branches; if restricted to the fields corresponding to one or more spinal segments the cord is at fault; if they chiefly affect one side of the body, the brain. The nature of the sensory phenomena has little value. Intense pain, for example, may be symptomatic of peripheral nerve disease as in some forms of neuritis, or of a degenerative process within the cerebrospinal axis as in tabes.

Irritative lesions cause disordered subjective sensations of heat, cold, formication, and the like—the paræsthesias—and pain of every variety as to kind and degree.

Destructive lesions, if they completely interrupt the sensory path, wholly abolish sensation in the parts of the body involved. A lesion of a peripheral sensory neuron in the course of the nerve gives rise to anæsthesia in the area of distribution of the nerve; a complete transverse lesion of the spinal cord gives rise to total loss of sensation of all parts below its level. Destructive lesions of the central nervous system do not however usually interrupt all the sensory conduction paths, and sensation may not be wholly abolished even in extensive disease. Sensation may be diminished or lost in all its phases as in complete transverse lesions of the cord, or there may be dissociation sensory paralysis as in certain diseases of the cord in which pain-sense and temperature-sense are abolished while tactile sensation remains unimpaired, or in some lesions of the cerebral cortex in which there may be a loss of the muscular sense and astereognosis—the loss of the ability to recognize an object placed in the hand—while other phases of sensation are fully preserved.

EXAMINATION OF THE PATIENT.

The Anamnesis.—An accurate history of the case is of the highest importance in disease of the nervous system. This must include in many cases the facts relating to the antecedents of the patient, which bear upon hereditary predisposition, as the occurrence of nervous or mental disease in the parents, children, or collateral members of his family. Peculiarities, idiosyncrasies, and psychoses are especially to be ascertained, often a matter of no little difficulty. A history of gout, alcoholism, or syphilis in a parent, when it can be obtained, may give the key to the situation.

The investigation of the personal history must bear upon any previous serious illness and its nature, whether nervous or not, and especially whether or not such an illness was of a similar nature to that from which the patient is suffering.

It may be necessary to follow in our investigation a chronological order, ascertaining whether or not nervous symptoms have occurred in infancy and childhood, such as convulsions, enuresis, night terrors. The period of school life is to be studied in obscure cases. The neurasthenic may have been bright and successful at school, but shy, retiring, and not disposed to make friends; the sufferer from *petit mal*, sometimes confused and forgetful; the hysterical girl, especially at puberty, nervous and emotional. The occupation is next to be considered. Is it one that involves continuous monotony, mental strain, extreme responsibility? Have there been prolonged or cumulative depressing emotions, disappointment, fear, sorrow, or grief? Wounds and injuries, alcoholism, and abnormal sexual matters, especially syphilis, are of etiological importance in many neurological cases. Severe infectious processes, particularly enteric fever, may have been the point of departure for visceral and vascular changes which after a time manifest themselves in the guise of nervous disease. Of special importance are such maladies in their relation to postinfective psychoses and neurasthenia. The part played by obscure toxæmias due to chronic gastro-intestinal or other visceral diseases in the etiology of certain spinal cord degenerations is not to be disregarded. Notwithstanding the number of points to be considered the value of the history cannot be measured by its length. On the contrary it is most important to briefly record only the facts which are pertinent and significant.

Status Præsens.—While investigation on every side is necessary for a full understanding of many nervous cases, yet there are certain special paths of approach which experience has taught us lead most directly to a diagnosis in the average case; in other words certain distinctly *neurological methods* of investigation. These methods may be grouped according to the character of the symptoms and signs that each brings into view, the most important being, (1) motor and (2) sensory symptoms; (3) cerebral symptoms, of which, on account of comprehensive and special characters, (4) aphasia requires separate consideration; (5) spinal symptoms in so far as they connect segments of the cord with particular regions of the body; (6) the reflexes; (7) electrical phenomena; (8) trophic disturbances; (9) pain and temperature; (10) muscular sense.

1. Motor Symptoms.

Paralysis.—Motor paralysis signifies impairment of some portion of the motor pathway. When partial it is to be distinguished from *akinesia*, common in states of mental stupor, and from incoördination, often mistaken by the patient and his friends for true weakness. The practical tests for muscular weakness consist, for the hand and forearm, in estimating the patient's "grip" as he squeezes the hand of the examiner, especially in comparing the grip of an affected hand with the other, which may be normal or less affected. Of mechanical devices the dynamometer of Math-

ieu is most commonly used. The power of arms and legs is tested by having the patient make various movements while the examiner, grasping the part, offers resistance.

A general surmise as to the location of the lesion (cerebral or spinal) causing the impairment of the motor path is made by observing whether the affected part is flaccid or spastic. Flaccidity nearly always denotes a lesion of lower motor neurons (ganglion cells of ventral gray horns, peripheral nerves with their terminals) as seen in poliomyelitis and neuritis, while spasticity signifies a lesion of central or upper motor neurons (cell-bodies of motor cortex, fibre tracts through subcortex, internal capsule, pons, medulla, ventral and lateral pyramidal tracts of spinal cord), as in old brain hemorrhage.

A notable exception to this broad rule is that in lesion of the spinal cord, complete or nearly complete transversely, especially one high up in the cord, the effect is as if all motor neurons below it were destroyed, *i.e.*, there is total flaccid paralysis below the level of the lesion. The explanations of this phenomenon are numerous but unsatisfactory. Another exception to this rule is readily correlated with it by bearing in mind that the superior motor neurons of the pyramidal tract are not wholly cerebral but have a spinal portion which is mostly contained in the lateral tract; hence it follows that a spinal palsy is spastic if the lateral tracts are involved.

To decide whether a member be flaccid or spastic, all the patient's active movements, including gait, are to be studied, as well as various passive motions which may suggest themselves to the examiner. His opinion will be rather one of judgment than of definite methods.

Monoplegia is a paralysis restricted to one member, whether this be disabled entirely or only in one group of muscles. Hemiplegia, or paralysis of one side of the body, is nearly always due to a brain lesion, and, when so, the upper face will be found unaffected or slightly affected, except in recent cases where the paralysis in the upper distribution of the facial nerve may be very distinct for a time. The slight implication of the upper face is characteristic of a long-standing cerebral hemiplegia. Diplegia—double hemiplegia—occurs particularly in childhood. Paraplegia is a symmetrical paralysis involving the upper or lower limbs, but when the term is used without qualification it refers to paralysis of the lower limbs. The term *brachial paraplegia* is employed to denote paralysis of the upper limbs; *crural paraplegia* that of the lower. It is generally a spinal palsy.

Contracture. — In paralysis of long duration contractures appear which are generally characteristic. Those which arise in spastic paralyses depend upon shortening of the paralyzed muscles, the stronger muscles contracting more than the weaker, and produce such postures of the limbs as are seen in hemiplegia (flexion of elbow, wrist and fingers, adduction of arm to chest, extension of the leg on the thigh, adduction of the knees, extension of the foot and inversion with plantar flexion of toes). According to some investigators the contractures of cerebral hemiplegia are the result of the greater paralysis in certain groups of muscles. The contractures in flaccid paralyses depend upon the unbalanced action of the opposing

sound muscles, as seen in the acceentuated wrist-drop and foot-drop of old peripheral neuritis, or depend upon the contraction of the paralyzed muscles themselves.

Convulsions and spasm (see Part III, p. 604).

Jacksonian epilepsy (see p. 605).

Athetosis or mobile spasm consists of irregular writhing movements, especially of the fingers but also of the arms and other parts. It is almost pathognomonic of the cerebral palsies of childhood, in which affections the symptom may mislead by being more prominent than the hemiplegia or diplegia which underlies it. Occurring in adult hemiplegics these movements are sometimes called *posthemiplegic chorea*, but are less prominent than the weakness and rigidity of the limb. Athetosis is usually aggravated by voluntary movements, as when the patient attempts to pick up a small object.

Tremor (see p. 608).

Fibrillary tremor or fibrillary twitching (see p. 609).

Tics.—Twitching simultaneous over a large area, inducing a purposive movement at intervals and habitually, is called a "tic." It is not a sign of any known lesion but is functional (a neurosis).

Ataxia.—In the course of investigation of motor signs the examiner may observe irregularity and uncertainty in various acts which require a degree of precision. Ataxia results from inharmonious action of muscle-groups even when disorder of motility, either excess or deficiency, is not present.

The defect is largely in the muscular sense, which is discussed in its relation to astereognosis. Yet the practical tests for the symptom are motor. In the arm ataxia is discovered by directing the patient to close his eyes and then with his index finger to touch the tip of his nose, or to meet the tip of the other index finger in sweeping the arms around horizontally in front; in the leg, by having him attempt to touch one knee with the heel of the other foot. If there be considerable ataxia the patient touches wide of the mark. Ataxia of the legs is better revealed in the patient's manner of walking, which is considered in connection with other disorders of gait.

2. Sensory Symptoms.

Studies of sensation involve a subjective element which makes them at best uncertain. Scientific methods aim to diminish this uncertainty by magnifying the objective element through the use of technical procedures which render the examiner less dependent upon the patient's statements. In children, and in stuporous and demented patients, the objective element alone is considered—a start, a vocal sound, or the withdrawal of a member when the patient is touched, pricked, etc.

Paræsthesia.—"Numbness and tingling," "pins and needles," "crawling sensations"—formication—and burning sensations are symptoms of sensory irritation. They are prominent in neuritis, and in spinal diseases which implicate the posterior nerve-roots (see also p. 598).

Delayed Sensation.—Recognition of any artificial sensation is, for the purposes of the clinician, instantaneous; if an interval occurs between the application of a stimulus and the patient's response to it, we speak of "delayed sensation," which is common especially in tabes dorsalis.

Pain.—Pain is a prominent symptom of many nervous diseases.

The objective study of sensation comprises the testing of the senses of touch—common sensibility—of pain, and of temperature. The muscular sense is of interest clinically in relation to astereognosis, and also in relation to ataxia of movement, which may arise from defect of the muscular sense.

In testing the sense of touch it is well to blindfold the patient, to take care that the surface examined shall not be chilled by exposure, and to touch the part with light pressure and without causing pain. The instrument most commonly used for this purpose is Carroll's æsthesiometer, but a tooth-pick or a feather will serve. The patient is directed to say "now" when the touch is felt; or to count successive touches a short distance apart, "one, two, three," etc., and the failure to note one or more touches will mark the boundary of an area of anæsthesia. Sensibility to touch is more acute on the back than on the front of the body. Loss of tactile sensibility, either total—*anæsthesia*—or partial—*hypæsthesia*—may be functional and a sign of hysteria, in which case it commonly affects half the body—hysterical hemianæsthesia—or a segment of one limb, or all of one extremity up to a certain level—"glove-anæsthesia" and "stocking-anæsthesia;" or anæsthesia and hypæsthesia may constitute a sign of organic nervous disease which is destructive in character or is at an advanced stage. In the case of hemihypæsthesia or hemianæsthesia, the hemorrhage or other destructive lesion may be in the posterior part of the internal capsule—where according to some anatomists sensory fibres are collected into a bundle (*carrefour sensitif*)—in the tegmentum of the pons, or in the spinal cord, provided one lateral half of the pons or cord be severed. In any of the cases mentioned the lesion is situated on the side opposite to that of the anæsthesia.

In testing the pain sense, a needle-point or one of the sharp points of the æsthesiometer is employed, and the skin is "pricked," not scored, with the instrument. Remind the patient that actual pain, not the mere sense of being touched, is to call forth his response; or instruct him to say "touch," or "pain," according as the one or the other sensation is excited by the sharp point.

The temperature sense is well studied by the use of two test-tubes of water, one heated to about 100° F. or above, the other cooled to 60° F. or lower, the tubes being applied alternately, and each being held in contact with the skin-surface for several moments, since recognition of heat or of cold is commonly less prompt than that of touch and of pain. The heat of the one tube should not be sufficient to burn, as that would introduce the factor of pain; yet practically this distinction is of little consequence, because the thermic sense and the pain sense, being conducted in adjacent tracts of the cord, are commonly abolished together. Ordinarily when tactile anæsthesia has been demonstrated in a certain area, we may expect to find thermo-anæsthesia and analgesia associated with it. But the converse of this does not always hold true; for over surfaces which betray no tactile anæsthesia, or at most only hypæsthesia, we may find areas of analgesia and thermo-anæsthesia. This is that *dissociated* sensory loss which is most common in syringomyelia, though other lesions of the central part of the gray matter of the cord may cause the phenomenon.

3. Regional Diagnosis of Cerebral Disease.

General Symptoms.—The general symptoms of intracranial disease—vomiting, headache, and optic neuritis—have little value in cerebral localization. Headache is more likely to be frontal in lesions of the fore-brain and occipital in those in or about the cerebellum, but this is not constant. Dense tumors of some size, well above the base of the skull, may yield a shadow on the X-ray plate.

Predominant mental symptoms are suggestive of lesion of the prefrontal lobes, particularly the left; but it must be remembered that after head injuries delirium, confusion, or stupor may ensue from shock, without reference to severity or site of the trauma, and moreover that demonstrable brain lesions are comparatively rare causes of insanity.

Paralysis.—Of motor signs indicating lesion of the precentral convolution, anterior to the fissure of Rolando, paralysis has the greatest localizing value. Paralyses in the distribution of cranial nerves, especially of several, commonly indicate lesion at the base of the brain. If a single cranial nerve is implicated, the lesion is probably outside of the central nervous system; if one arm or leg is paralyzed, a cortical lesion should be suspected, and this is rendered probable if the paralyzed part is the seat of clonic spasm. Paralysis of the face indicates lesion in the lower third of the Rolandic cortex; paralysis of an arm or leg, lesion of the middle or upper third respectively.

Astereognosis.—Pure motor phenomena point to a lesion anterior to the fissure of Rolando; if the lesion be posterior to this fissure (postcentral) the motor signs are likely to be associated with the phenomenon called astereognosis, which becomes more prominent as the parietal lobe is encroached upon. By study of the "stereognostic sense" which is the physiologic process by which solid objects are recognized by contact, neurologic diagnosis has made a distinct advance. Astereognosis, or want of this sense, may be diagnostic of lesion of the superior parietal lobule. To test for this phenomenon it is well to study separately the several processes by which normally the hand recognizes the shape and size of objects, especially the "spacing sense," the sense of position, and the pressure sense, the last two of which are the chief components of the muscular sense.

The **"spacing sense"** is tested by touching the skin at two points simultaneously, as with the two arms of the æsthesiometer, and observing how near together they may be while still recognized as two points. The examiner compares his results with those obtained in a normal subject.

The **sense of position** is studied by asking the patient (blindfolded) to tell where his hand or foot is, after the examiner has quietly placed it in a particular attitude, or to imitate with one limb an attitude given to the other by the examiner.

The **pressure sense** is tested by blindfolding the patient, placing his hand supine upon a table, and laying in his palm, one after another, small objects identical save in their weight, which is graded in a series. For this purpose cartridges filled with layers of cotton and regulated numbers of buckshot may be used. The main test, which reveals astereognosis directly if it be at all pronounced, consists in handing

the patient various common objects,—watch, spool, block of wood, penknife,—each of which he essays to name or to describe.

Deafness, in the absence of disease of the external, middle or internal ear, may be due to lesion of the first or second temporal convolution, particularly that of the left side.

Blindness without demonstrable cause in the eye may be due to lesion anywhere in the course of the optic nerves, tracts or "radiations" as far as the cunei lobes, which face one another across the great longitudinal fissure in the occipital lobe. Unilateral blindness of both eyes and in the same side of each eye (lateral homonymous hemianopsia) indicates that the lesion is unilateral, that it is back of the optic chiasm and is on the side opposite to that on which the patient's vision has failed—that is, on the same side as the blind half-retina. To determine how far back of the chiasm such a lesion is we must rely on signs and symptoms arising from involvement of contiguous structures, especially (in lesions at the base of the brain) the cranial nerves, which are spared in lesion of the optic radiations, or of the cuneus—subcortical or cortical lesions. A theoretically positive means of distinguishing basal from cortical lesions causing hemianopsia is WERNICKE'S PUPILLARY-INACTION SIGN, which consists in the absence of the light reflex of the iris when only the blind half of the retina is illuminated. The finding of this condition points to a basal lesion, *i.e.*, at or below the optic thalamus and external geniculate body, for contraction of the iris is a function of the third nerve, and no part of this nerve extends above the "primary optic centres," which are at the base of the brain.

Partial loss of vision, not accounted for by eye disease, may be due to lesion of the angular gyrus, visual acuity—macular vision—being impaired; or it may be due to lesion in front of the optic chiasm, in the angle between the optic nerves, where by interfering with the internal fibres of each nerve it causes blindness of each inner (nasal) half-retina, a condition called (from the blind half-fields) temporal hemianopsia, which is pathognomonic of lesion in the situation described.

Symptoms of Cerebellar Disease.—The cerebellum is to the clinician chiefly an organ of coördination, and this function resides mainly in the middle lobe. The cardinal signs of cerebellar disease are *nystagmus* and a peculiar ataxia which gives a staggering character, or *titubation*, to the patient's gait. This ataxia disappears when the patient lies down, and the knee-jerks are often preserved. Neoplasms beneath the middle lobe of the cerebellum are likely to cause this form of ataxia together with external ocular palsies from pressure upon the nuclei of the third and fourth nerves beneath the quadrigeminal bodies. A tumor arising from these bodies can hardly be distinguished from cerebellar tumor implicating the vermis.

The Internal Capsule.—Of the great interior structures of the brain only the posterior limb of the internal capsule has functions so definite that certain symptoms may be referred to it. Sudden hemiplegia, with hemianæsthesia and hemianopsia, is generally indicative of lesion in the internal capsule, since this complex of symptoms from cortical or even subcortical lesion could be induced only by uncommonly extensive damage. The

symptoms referable to single minute destructive foci in the posterior limb of the capsule, from the "knee" backwards, are, so far as is known, (1) paralysis of the face from above downwards, (2) of the arm and (3) of the leg, also from above downwards, (4) anæsthesia of varying extent up to hemianæsthesia, which probably indicates destruction of the posterior third of the posterior limb, (5) hemianopsia.

Cerebral Ganglia.—Of the great cerebral ganglia none has an independent symptomatology. Lesions affecting the corpus striatum cause predominant motor signs because of pressure upon the motor bundles of the capsule, while affections of the optic thalamus commonly cause hemianæsthesia from pressure upon the posterior fibres of the capsule—*carrefour sensitif*—or destruction of sensory fibres within the thalamus and often hemianopsia from involvement of the optic radiations, which are collected into a bundle posterior to the capsule and enter the optic thalamus. Mobile spasm or athetosis, associated with these paralyses, is in favor of thalamic lesion. Weakness of the articulatory muscles resembling bulbar paralysis, but not due to lesion of the medulla oblongata, is called pseudobulbar paralysis. It is most often due to multiple hemorrhages or softening in the outer part of the lenticula.

Lesions of the corpus callosum are revealed by disturbance of the functions of surrounding parts, notably of the motor zone,—as shown by early epileptic seizures, by paralyses, and symptoms referable to the prefrontal region. From the latter arise the pseudoparetic mental states which are characteristic of callosal lesion.

4. Aphasia and Other Defects of Speech.

Though endowed with a normal brain, the individual born deaf and blind becomes an imbecile by deprivation of the sense-impressions out of which knowledge grows, unless he be trained like Laura Bridgman through the touch-sense. The cochlea, the retina, etc., begin the transformation, from mere contact with the external world, into the higher special sense-impressions. These, carried by their separate paths to the cortex, are elaborated in the special-sense centres into perceptions of things. Roughly speaking, each cortical centre is opposite the organ of that sense. Taking one sense, vision, rays of light from an object, for example a cow, received by the retina are carried through the visual system to the cuneus as sensations of form, color, etc. Thence passing still higher, in the angular gyrus is formed a visual image of a cow—object-seeing—and this is associated with an image of the word cow written or printed—word-seeing. Lesion of angular gyrus then does not cause ordinary blindness—as lesion of the cuneus does in one half-field—but loss of these visual images, so that the patient seeing a cow can hardly tell it from a horse—object mind-blindness; and seeing the word cow fails to get the meaning from it, as if it were a foreign word—word-blindness. In like manner, close to the auditory centre is a higher centre for the formation of auditory images, by which a peculiar sound is identified, for example, as the lowing of a cow—object-hearing—and by which the spoken word cow is recognized as the name of that animal—word-hearing. With a lesion then in the first temporal

convolution, sparing the main auditory centre, so that the patient is still capable of hearing noises, there may be loss of these auditory images with consequent object-deafness and word-deafness; sounds and words heard are meaningless.

To speak of a cow it is necessary to recall the word cow. Many persons are likely to recall a word as it sounds; some as it appears written; but most revive it in both ways, so that impairment of either the auditory or the visual word-image interfering with the recollection of words causes aphasia, in the one case, from lesion of the angular gyrus—visual or optic aphasia; in the other, from lesion of the first temporal convolution—auditory aphasia. In either case due to a lesion of a sensory centre it is spoken of as sensory aphasia; and because the essential defect is inability to recollect words (verbal amnesia) both are included under the term amnesic aphasia. The act of speaking involves several groups of muscles, and is interfered with, therefore, in various forms of paralysis. In bulbar paralysis, the lips, tongue, etc., becoming atrophied and paretic, there is defect of articulation, incidentally; and in the similar paralysis from cerebral lesion (pseudobulbar paralysis) the patient may be inarticulate. In like manner lesion in the cortical centres for the lips, tongue, etc., at the foot of the motor zone, cripples the speech just as lesion in the leg centre causes limping; so here on the emissive side of the speech-process there is set apart a higher centre for the fine adjustment of movements in uttering words, and for the memory of these movements. This is Broca's centre, in the posterior part of the third frontal convolution. By lesion here, the muscles of articulation still intact, the patient loses his motor memories and his power to utter words. This is motor aphasia—or aphemia.

Parallel to these defects of articulation are defects in the act of writing which has its higher centre in the second frontal convolution, related to the arm centre as Broca's is to the centres for the tongue, lips, etc. Lesion in the writing centre causes motor agraphia, even though the arm be still useful otherwise. In lesion of the angular gyrus, as the appearance of words is forgotten, writing is imperfect; there is sensory agraphia. In reading aloud, the image of the printed word must be conducted from the angular gyrus to Broca's centre, there to be matched with the motor image used in uttering the word; and the utterance must be guided, too, by the auditory image conducted from its centre. For this purpose Broca's centre is connected with the others by tracts of fibres which being damaged, particularly in the insula—island of Reil, there is interference with the conduction referred to, and hence, with reading aloud and with similar uses of speech, conduction-aphasia. For perfect speech all the centres must act in unison through conduction-paths connecting each centre with the rest, and considering such multiple connections it is evident that aphasia of some kind may result from lesion at any point within a wide area. This "zone of language" is nearly coextensive with the distribution of the middle cerebral artery, and aphasia is generally a consequence of apoplexy from this vessel, commonly in association with hemiplegia. This same region, acting as a unit, forms a complete image not alone of the word but of the object also, as it looks, sounds, feels, smells, tastes—in short, a concept of the object; so that this is a concept area (Mills).

Aphasia being a curtailment of the power to comprehend as well as of the power to emit language, spoken, written or by signs—pantomime—care and system in testing for it are very important. "Impediments" of speech, mechanical imperfections of the vocal organs, are first to be eliminated by examination of the mouth, throat, and nasal cavities. In cleft palate, hypertrophic rhinitis, and in tongue-tie, the difficulty is mainly in the enunciation of consonants, such as m, n, b, etc.

Dysarthrias, from paralysis or defective innervation of the muscles of articulation, are to be recognized partly by finding additional signs of cerebral paralysis or other organic nervous disease, and partly by special characters of the speech in certain affections. Somewhat suggesting mechanical impediment is the speech of bulbar paralysis, marked as it is by labored pronunciation of consonant-sounds.

Elision of syllables by running words together, may be observed in hereditary ataxia, in which disease speech is at the same time monotonous. These two characters belong also to the speech of general paresis, forming with the difficulty of enunciating the r's and l's, as in "artillery," and with its tremulous, measured drawl, the peculiar "paretic speech" which is one of the cardinal signs of this disease. The measured character of such speech exists in purer form—scanning—in disseminated sclerosis.

Ordinary stuttering is a pure neurosis. It manifests itself by spasmodic halting in attempts to utter certain words, usually those beginning with consonants.

In differentiating aphasia from other speech defects the greatest difficulty arises in the case of actual mental loss—dementia—which indeed may coincide with aphasia, as in hemiplegia and senility, or may have aphasia for an episodic manifestation, as in paresis.

The stubborn speechlessness frequently met with in paranoia and melancholia is nearly always accompanied by other signs of negativism, as refusal of food and resistance to the attentions of the nurse. Hysterical aphasia is intermittent and its victim exhibits the stigmata of the neurosis.

In testing an apparent aphasic it is well to begin on the sensory side, ascertaining whether the centres for word-hearing and word-seeing are impaired. A number of common objects may be placed before the patient who endeavors to pick out those named in turn by the examiner and then to select from a list of names on paper, that of the object selected by the examiner.

As the purpose is to determine the clearness of word-images, these simple tests are essential; but the examiner may progress to words and sentences of any complexity. Rarely being complete, aphasia is often betrayed by persistent, helpless misapplication of words, the patient saying or writing for instance "dog" when a hat is showed to him and its uses demonstrated by him. Paraphasia and paragraphia are forms of aphasia rather characteristic of sensory aphasia.

To test a patient's emissive power of language, that is, to discover motor aphasia and agraphia, objects are shown to him, and he endeavors to utter and write their names. Simple acts performed in the patient's presence are described by him both orally and in writing. In motor aphasia "recurring utterances" are common, a patient repeating "any one any" or other meaningless phrase on all occasions when attempting to talk.

Even after demonstrating that a patient hears, reads, utters, and writes words correctly, and thus that the widely separated cortical speech-areas in the first and second left temporal convolutions, the angular gyrus, the third frontal and the second frontal convolutions are probably intact, together with the subcortical region corresponding to each centre, we may still find that the patient is much crippled in the use of language. In such a case we conclude that some of the fibres connecting these cortical centres with one another are cut off, and that we are dealing with a case of "conduction aphasia" or "transcortical aphasia." The prominent features of this form are paraphasia, paralexia, etc., so extreme that the jumbling of words and syllables is spoken of as "jargon-speech."

So entangled in these "association-systems" are all the cortical centres that speech-defect from cortical lesion always betrays some features of conduction aphasia. If our tests reveal uncomplicated word-deafness or word-blindness, or simple loss of the power of utterance, we recognize that the lesion is deep in the brain, beneath the level of association-systems, where the fibres radiating to or from the particular centre are bundled together. This is called "subcortical" or "pure" aphasia, and yet some evidence exists that this form of aphasia may result from cortical lesions.

As an auxiliary test for this form, the study of pantomime is of value. In pure motor aphasia, for instance, the patient though speechless as regards utterance, may when asked how old he is, open and shut his hand the proper number of times. In ordinary motor aphasia this is impossible.

5. Spinal Localization.

One of the consequences of modern clinico-pathological study is the tendency to interpret nervous symptoms and signs in relation to anatomical structure, rather than in relation to empirical disease forms. This tendency in the field of brain disease has created cerebral localization; and it has affected our conceptions of spinal disease to the extent that we speak less of "locomotor ataxia," of "spastic paraplegia" or of "progressive muscular atrophy" as disease entities than as dominant symptoms of various lesions affecting certain structures of the spinal cord. Assuming in this connection that the symptoms in a given case are of spinal origin, we infer from "ataxia" of a limb that the dorsal column of the cord is affected; from spasticity with increased reflexes, the pyramidal tract; from atrophy, the ventral horns of the gray matter; from anæsthesia, the dorsolateral column again: from loss of pain- and temperature-sense without anæsthesia—dissociation of sensation—the central part of the gray matter; from pain, the dorsal roots. Then we endeavor to determine the lesion which has caused the particular symptom-complex which confronts us by bringing to bear our knowledge of the natural history of nervous disease and by collating the spinal symptoms with any cerebral manifestations which may be present. By this method we may find that our "locomotor ataxia" case is really one of combined degeneration of the cord or one of paresis, and that "progressive muscular atrophy" is symptomatic of syringomyelia or of tumor.

Spinal localization in the ordinary sense, however, relates to diagnosis of the level of a lesion in the cord. It is based upon our accumulated knowledge of the motor sensory reflex and sympathetic control exercised by each segment of the spinal cord over a corresponding segment of the body.

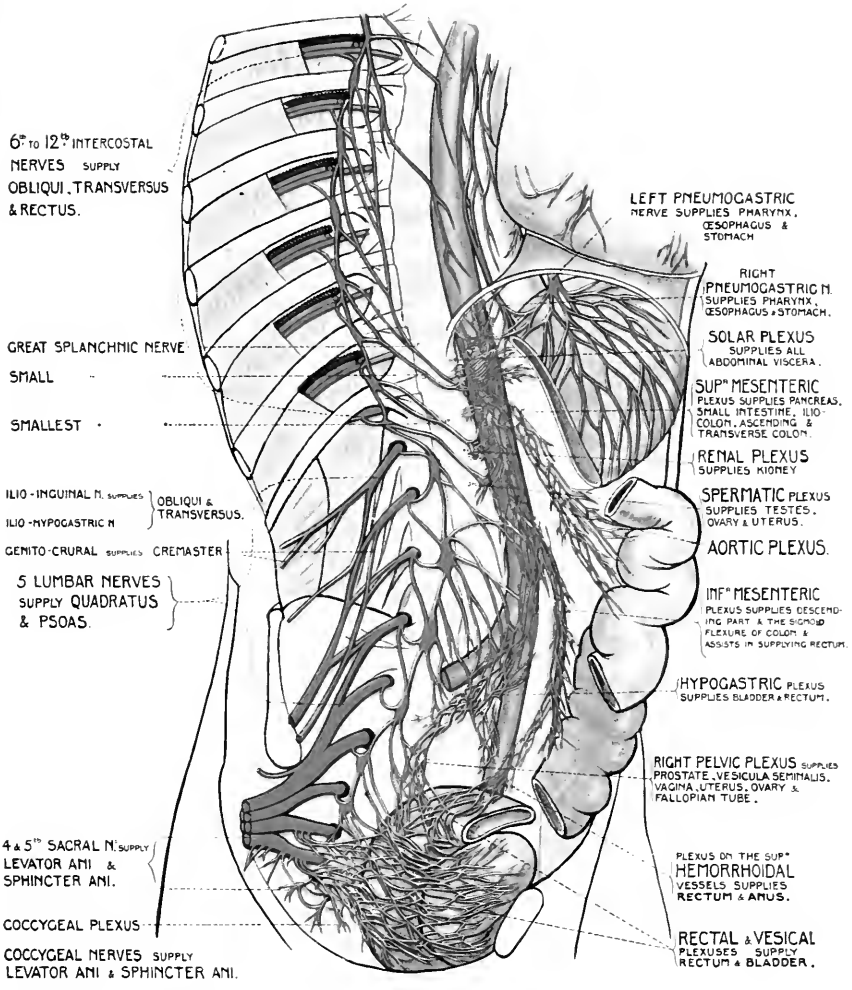
Injury to the spinal cord at any point involving the motor tracts—unless it be completely severed—causes paralysis, with increase of reflexes, below that point; but at the level of the lesion we are likely to find the reflexes abolished. We commonly find also anæsthesia covering the body below this level if the lesion is grave, and the upper limit of anæsthesia, with the zone of absent reflexes coinciding, is the best index to the level of the spinal lesion. If the lesion affects one lateral half of the cord the above principles still apply, but the disturbance of sensation, except of the sense of position, is found on the side opposite to that of the lesion and to that of the motor symptoms—Brown-Séquard's paralysis, although even in this form tactile sensation is often preserved.

The level thus ascertained marks the relative position of the lesion, but its actual position in the spinal column will be found above this, generally a distance of about three spinal segments. A narrow zone of anæsthesia is usually present in Brown-Séquard's paralysis on the side of the lesion, and at its level and above this may be a narrow zone of hyperæsthesia. Such an anæsthetic zone occurring independently points to a lesion outside the cord substance and involving spinal roots of at least two segments. Sensory loss from injury to the cord proper or the posterior roots is distributed in horizontal bands about the trunk and longitudinal bands in the limbs, irrespective of the distribution of the nerves—segmental anæsthesia.

The clinician should be able to conclude off-hand from atrophy of the shoulder, or loss of reflexes in that region, that the upper cervical region is affected when the symptoms are of spinal origin; from such symptoms affecting the forearm and hand, that the lesion is lower down in the cervical swelling; from loss of knee-jerk, that it is in the lumbar, and from loss of control of sphincters, in the sacral region; but for finer deductions it is well to record the findings in a particular case, and then interpret them by reference to the tables and diagrams upon pp. 326, 327 and 328.

Combined Degenerations.—"Typical cases" are as narrow summits in the great ranges of disease. From each summit the symptomatology and pathology form a downward slope, by which that disease merges with one or more of its neighbors. Of lateral sclerosis very few absolutely pure cases have been reported. On the one hand, in cases that seem like pure lateral sclerosis, there is nearly always insidious degeneration in the ventral gray horns—chronic poliomyelitis; or the latter disease after a course of years may take on spastic symptoms because the pyramidal tracts are invaded, that is, degeneration beginning in either motor neuron tends to progress to the other.

In some cases the affection of superior and inferior motor neurons is simultaneous, progressive muscular atrophy and spastic paraplegia developing *pari passu*. Such cases constitute amyotrophic lateral sclerosis. In them the bulbar part is prominent and degeneration may extend even to the cortex, mapping out the motor zone, for amyotrophic lateral sclerosis is a disease of the whole motor system.


6th to 12th INTERCOSTAL NERVES SUPPLY OBLIQUI, TRANSVERSUS & RECTUS.
GREAT SPLANCHNIC NERVE
SMALL
SMALLEST
ILIO-INGUINAL N. SUPPLIES
ILIO-HYPOGASTRIC N
OBLIQUI & TRANSVERSUS.
GENITO-CRURAL SUPPLIES CREMASTER
5 LUMBAR NERVES SUPPLY QUADRATUS & PSOAS.
4 & 5th SACRAL N. SUPPLY LEVATOR ANI & SPHINCTER ANI.
COCCYGEAL PLEXUS
COCCYGEAL NERVES SUPPLY LEVATOR ANI & SPHINCTER ANI.
LEFT PNEUMOGASTRIC NERVE SUPPLIES PHARYNX, ŒSOPHAGUS & STOMACH
RIGHT PNEUMOGASTRIC N. SUPPLIES PHARYNX, ŒSOPHAGUS & STOMACH.
SOLAR PLEXUS SUPPLIES ALL ABDOMINAL VISCERA.
SUP. MESENTERIC PLEXUS SUPPLIES PANCREAS, SMALL INTESTINE, ILIO-COLON, ASCENDING & TRANSVERSE COLON.
RENAL PLEXUS SUPPLIES KIDNEY
SPERMATIC PLEXUS SUPPLIES TESTES, OVARY & UTERUS.
AORTIC PLEXUS.
INF. MESENTERIC PLEXUS SUPPLIES DESCENDING PART & THE SIGMOID FLEXURE OF COLON & ASSISTS IN SUPPLYING RECTUM.
HYPOGASTRIC PLEXUS SUPPLIES BLADDER & RECTUM.
RIGHT PELVIC PLEXUS SUPPLIES PROSTATE, VESICULA SEMINALIS, VAGINA, UTERUS, OVARY & FALLOPIAN TUBE.
PLEXUS ON THE SUP. HEMORRHOIDAL VESSELS SUPPLIES RECTUM & ANUS.
RECTAL & VESICAL PLEXUSES SUPPLY RECTUM & BLADDER.

Connection between sympathetic nerves supplying viscera and spinal nerves supplying muscles of abdominal walls.

On the other hand degeneration in the lateral columns may be accompanied by dorsal degeneration. Such implication of *superior motor* neurons with *inferior sensory* neurons in combined degeneration suggests a local, extraneous cause, acting upon the lateral and posterior columns simultaneously. The mechanism for this could be the marginal system of arteries carrying some toxin into these portions of the white matter, and the lesion might be expected to spread around the arterioles from the very margin of the cord. These conditions are clearly present in some cases of ergotism, pernicious anæmia, etc. An acute diffuse dorsolateral degeneration may be found at autopsy, though commonly no symptoms have been observed in life. In various undetermined toxæmias (Putnam and E. W. Taylor) a subacute degeneration is established, partly diffuse, but also partly systematic, as in time it enters the course of ascending and descending tracts. This subacute combined degeneration is clinically distinct from the other types (Russell, Batten and Collier). Possibly similar in origin but appearing as a pure combined system disease (Strümpell), the chronic form, posterolateral sclerosis, has been recognized longer. It is manifested clinically by paraplegia from the pyramidal tract lesion, with ataxia from the lesion in the dorsal columns—ataxic paraplegia of Gowers—but without lightning pains or other sensory phenomena and without eye-symptoms, because the sensory root-zones and roots—including the optic nerves—are spared. For this last reason, too, the reflexes are preserved in posterolateral sclerosis; by the degeneration of the pyramidal tracts they are usually increased and the legs made spastic. At a late stage the root-zones may be invaded and reflexes impaired until the case appears like one of simple tabes dorsalis, only an autopsy revealing the combined lesion.

A combined sclerosis is the commonest spinal lesion of paresis.

6. The Reflexes.

Every segment of the spinal cord contains not only centres for certain groups of muscles but also for reflex movements. The reflex starts in an impulse arising from the stimulation of a sensory nerve. It is transmitted to a centre in the cord and passes by way of the processes of the sensory cell-bodies to the neurons of the corresponding motor centre, in which it originates a motor impulse which in turn passes by way of the motor nerve to the muscle-fibres supplied by the nerve. This complete path, made up of centripetal or sensory fibres with their cell-bodies and correlated cell-bodies with their centrifugal or motor fibres, is called a reflex arc. The sensory impulse may be transmitted to centres at higher or lower levels and excite several motor impulses, thus producing a complicated reflex arc. The cord segments are connected with fibres from the cerebrum having the function of inhibiting the reflex. If these fibres are irritated the reflexes are impaired from abnormal inhibition; if they are destroyed the reflexes are exaggerated. If the arc is interrupted either in its afferent or efferent limb or in the centre the reflex is lost.

Involuntary contraction of muscles aroused by a sensory impression upon related parts is a reflex in the ordinary sense. For fine deductions the muscles themselves must be observed. The quadriceps cruris, for

example, may be seen to contract on tapping the patellar tendon, even when no motion of the leg occurs, and under such circumstances the "knee-jerk" cannot be said to be abolished; but ordinarily we recognize reflex response in muscles by a characteristic motion imparted to a member, as the kicking movement of the leg which is regarded as a measure of the knee-reflex.

Absence of the usual motor response, of the knee-jerk for example, or its diminution or exaggeration, are the matters to be attended to in the study of most reflexes, particularly the "tendon-reflexes." This is true also of most of the superficial—skin—reflexes, though in certain of them the character of the motion elicited is significant; thus with the plantar reflex, flexion of the toes is normal, while extension—Babinski reflex—indicates lesion of the pyramidal tract of the corresponding side, extension of the toes being equivalent to exaggeration of other reflexes. In a third group, the so-called periosteal reflexes, any motion of the member establishes the presence of the reflex, as in the case of the scapulohumeral, the motion of which may be external or internal rotation, and ad- or abduction of the upper arm, according as to which of the muscles attached to the scapula are most actively excited when this bone is jarred by tapping at a spot where it is bare save of periosteum and skin.

A reflex must be fairly constant and discernible in the normal subject to give much significance to its alterations, particularly to its absence. Many reflexes are of minor clinical importance because they are present in only a small percentage of normal subjects and then are not pronounced, the ulnar for instance. Reflexes of the lower extremity are on the whole more important than those of the upper, and the knee-jerk is preëminent in this respect.

The reflexes of the upper extremity being inconstant, absence of any one of them signifies little; exaggeration of one has a certain value; and even the marked presence of a number of them in a patient has something of the import of exaggeration of other reflexes.

Knee-jerk or Patellar Tendon Reflex.—To elicit the knee-jerk the leg is rendered passive by crossing the knee over its fellow, or by supporting it on the examiner's forearm passed under the patient's knee and braced by the hand placed upon the other knee, or by having the patient while recumbent draw up his knee into an easy position with all muscular tension on his part withdrawn; the patellar tendon well below the knee-cap is then struck a firm, quick blow with the ulnar edge of the hand or with a percussion hammer.

The knee-jerk should never be declared absent until Jendrassik's method of reinforcement has confirmed the result. This is applied by directing the patient to hook his hands together and to keep them so while tugging at them as if to pull them apart. It is customary for the examiner to count "one, two, three" after instructing the patient to "pull hard" at "three," the tap on the tendon being made at about "four."

The signs + for increased and — for diminished knee-jerks are commonly employed; and, in writing, "kj" for the reflex itself is allowable.

The knee-jerk being due to contraction of the quadriceps cruris muscle, the essential phenomenon may be induced by tapping the

muscle itself just above the patella, especially if the latter is pressed downward by a finger laid along the upper edge of the bone and this finger is then tapped with the hammer.

FIG. 133.—Method of testing patellar reflex.

FIG. 134.—Achilles tendon reflex.

Babinski Reflex.—In testing for the Babinski reflex the examiner supports the patient's ankle with his left hand and strokes the sole of the

FIG. 135.—Plantar flexion.

foot with any object which makes a distinct sensory impression—a somewhat sharp point being necessary when the skin is thick—at the same time noting the movement of the toes, which in all normal persons past the age

of infancy is plantar flexion. Extension (dorsiflexion) of the toes, particularly of the big toe, elicited in this way constitutes the Babinski reflex, which is a most important sign of involvement of the pyramidal tracts.

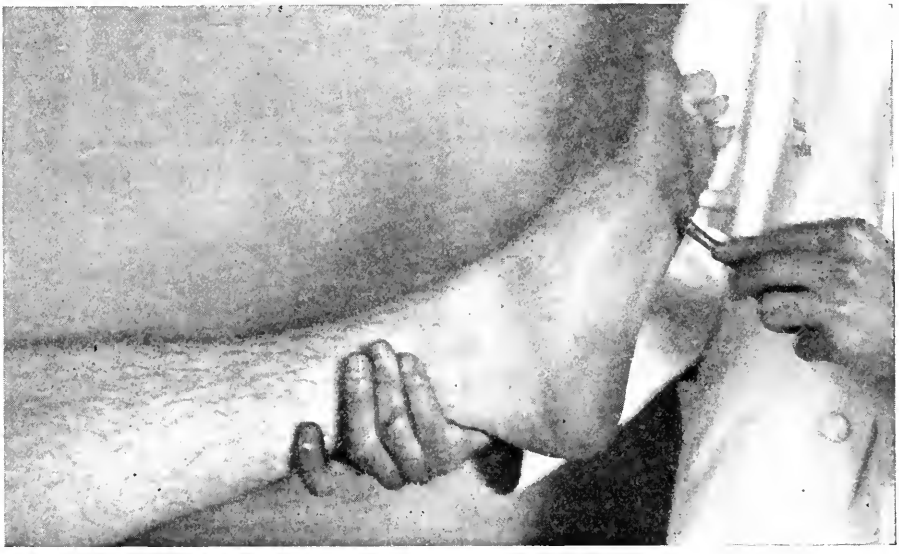


Fig. 136.—Babinski reflex (dorsiflexion of the toes).

Ankle Clonus.—This phenomenon usually accompanies a considerably increased knee-jerk, and has a similar significance. To test for it, the whole leg should be relaxed—best by having the patient supine. The

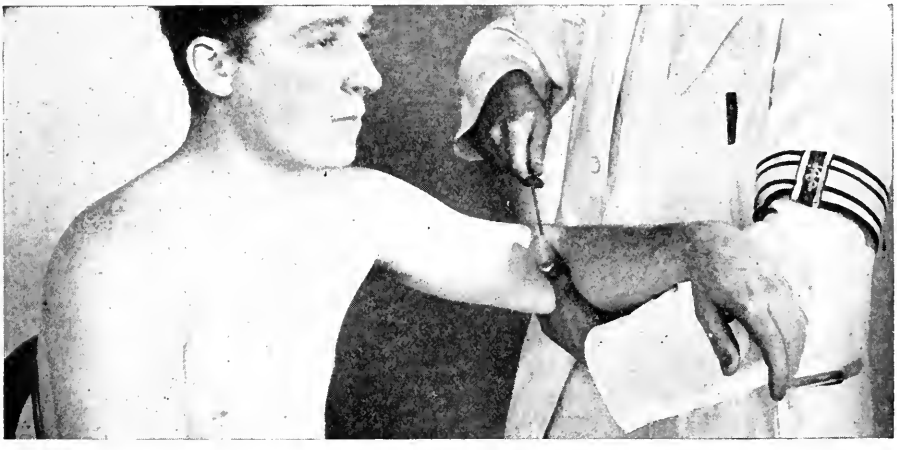


Fig. 137.—Biceps reflex.

examiner's left hand supports the leg, and his right, clasping the patient's foot, presses it upward, when, if clonus is present, the foot is pushed back against the hand in a series of jerks which are due to clonic spasm of the (soleus) muscle.

Achilles Jerk.—A single reflex-contraction of the calf muscle may be induced by tapping the tendon, which the examiner has rendered tense by pressing the foot upward. This reflex is called the Achilles jerk and is an index of the condition of the sciatic nerve and corresponding segments of the spinal cord. It is best obtained by having the patient kneel upon a chair while the examiner taps the Achilles tendon.

The Abdominal Reflex.—This reflex, quite a constant one, is elicited by stroking the side of the abdomen. The ensuing contraction is widespread over this region.

The Cremasteric or Inguinal Reflex consists in a drawing-up of the scrotum and testicle on stroking the inside of the thigh.

The Epigastric Reflex.—On stroking along the costal margin the muscles over the pit of the stomach contract.

Among the reflexes of the upper extremity, the **Radial**—a periosteal reflex—is elicited by tapping above the styloid process of the radius, and consists mainly in flexion at the elbow-joint. The nearly identical motion

FIG. 138.—Triceps reflex.

of the **Biceps Reflex** arises when the tendon of this muscle is tapped at the bend of the elbow. In testing the last two reflexes the examiner places his forearm under that of the patient, in order to relax the latter.

To elicit the **Triceps Reflex** the patient's upper arm is given a fixed support on the examiner's wrist or on a chair-back, when tapping above the olecranon causes an outward jerk of the forearm.

Plantar Reflex.—Produced by tickling the sole of the foot. It consists, when fully developed, of sudden withdrawal of the foot by flexion at the hip and knee, dorsal flexion of the ankle and plantar flexion of the toes. The movement in undeveloped cases may consist of sudden plantar flexion of the toes. It occurs in normal conditions, but in varying degrees. There are those who have the power to voluntarily prevent it. This reflex is exaggerated in neurasthenia, hysteria and other functional diseases of the nervous system, and may be associated in extreme cases with general convulsive movements or may be crossed,—that is, it may occur on the opposite side, as well as on the side tickled. It is also increased, but usually to a moderate extent only, in organic disease of the central nervous system. It is as a rule abolished in the affected side in hemiplegia and invariably absent in destructive lesions involving the sensory nerves of the legs.

Other reflexes of minor clinical importance are:

The Supra-orbital Reflex.—Produced by a sharp tap upon the trunk of the supra-orbital nerve, it consists of slight, momentary contractions of the orbicularis palpebrarum, especially in its external half. It is absent in destructive lesions of the supra-orbital nerve and in peripheral facial palsy.

The Malar Reflex.—Not usually present in normal conditions, but caused in recent facial paralysis of peripheral origin by percussion over the malar bone. It consists of contraction of the elevator of the angle of the mouth and movements of the ala nasi.

The Chin Reflex.—This phenomenon is elicited by tapping upon a small flat object, as an ivory paper cutter or a tongue depressor, laid upon the lower front teeth, or the finger laid upon the protuberance of the chin when the mouth is open and the jaw relaxed and drooping. The response consists in a sharp upward movement of the jaw. It may be present in nervous conditions, as hysteria, and in cachectic states.

Fig. 139.—Paradoxical reflex.

The Femoral Reflex.—Not present in health. It is produced in transverse lesions of the spinal cord above the level of the eighth dorsal segment by irritation of the anterior surface of the upper part of the thigh, and consists in plantar flexion of the toes and extension of the foot.

Sinkler's Toe Reflex.—Produced by sudden forcible flexion of the great toe. It consists in forcible flexion of the knee and hip and is met with in spastic conditions arising In spinal disease, as spastic paraplegia.

Gowers's Front Tap.—The leg being slightly flexed, a blow is struck upon the tibialis anticus muscle. Plantar flexion of the toes occurs in a considerable proportion of normal persons, many neurasthenic and hysterical individuals, and not at all in tabes.

Paradoxical Reflex.—Caused by sudden shortening of the tendon; elicited by deep pressure upon the calf muscles, and consisting in extension—dorsiflexion—of the toes, especially the great toe. It is regarded as a sign of irritation or early organic affection of the motor pathway.

Oppenheim's Reflex.—Dorsiflexion of the toes and foot upon forcibly stroking the skin along the inner border of the tibia.

Pflüger's Laws.—1. The reflex occurs upon the same side of the body as that to which the irritant is applied and in muscles whose motor nerves arise from the same segments of the cord. 2. If the reflex occurs on the opposite side, only the corresponding muscles contract. 3. If the reflexes are unequal on the two sides, the stronger are on the side upon which the

irritation has been applied. 4. When the reflex extends to other segments the direction of the extension is toward the medulla. 5. All the muscles of the body may yield reflexes.

Kernig's Sign.—Not a true reflex but conveniently described in this connection. Normally the leg may passively be fully extended on the thigh, when the latter is at right angles to the long axis of the trunk, as when the patient sits upon the edge of the bed with his legs hanging down, or has the thighs flexed when in the recumbent posture. The extending force must be moderate and gradual. Resistance and pain are developed at an angle between 95° and 135°. This sign occurs in acute meningitis, especially cerebrospinal fever, when collapse symptoms are absent, but is not constant in tuberculous meningitis. It has been variously ascribed to irritation of the meninges and posterior nerve roots, irritative lesions of the pyramidal tract, intraventricular pressure, and cerebellar irritation. It

Fig. 140.—Oppenheim's reflex.

occurs in various acute diseases in young children and very rarely in adults in enteric fever. It may be simulated in old age, disuse of the lower limbs, arthritis, sciatica, and contractures.

Brudzinski's Sign.—If the head is flexed upon the chest, flexion of the legs at the hips and knees occurs and flexion of one thigh upon the trunk causes a movement of the same kind on the other side.

7. Electrodiagnosis.

For diagnostic purposes the galvanic battery is more important than the faradic; but each gives considerable information as to the cause and character of motor paralysis or the variety of muscular atrophy present, and the prognosis in paralysis and atrophy of certain kinds.

The electrodes, covered with absorbent cotton and wetted, are placed upon the patient's bare skin, one at some "indifferent" point, as the back of the neck, the other upon the part to be examined—motor point of the muscles or the nerve-trunks. With a faradic current thus applied, on opening the circuit a quick contraction of the muscles ensues in the region of the distal electrode, whether this be positive (the anode) or negative (the cathode); but if the interruptions are rapidly repeated the muscle is thrown into a tetanic state. If these muscles be the seat of paralysis from lesion of the inferior motor neuron—poliomyelitis, neuritis, etc.—or if they be atrophied, their response to the faradic current is diminished in a degree which, after some experience, can be estimated by the examiner.

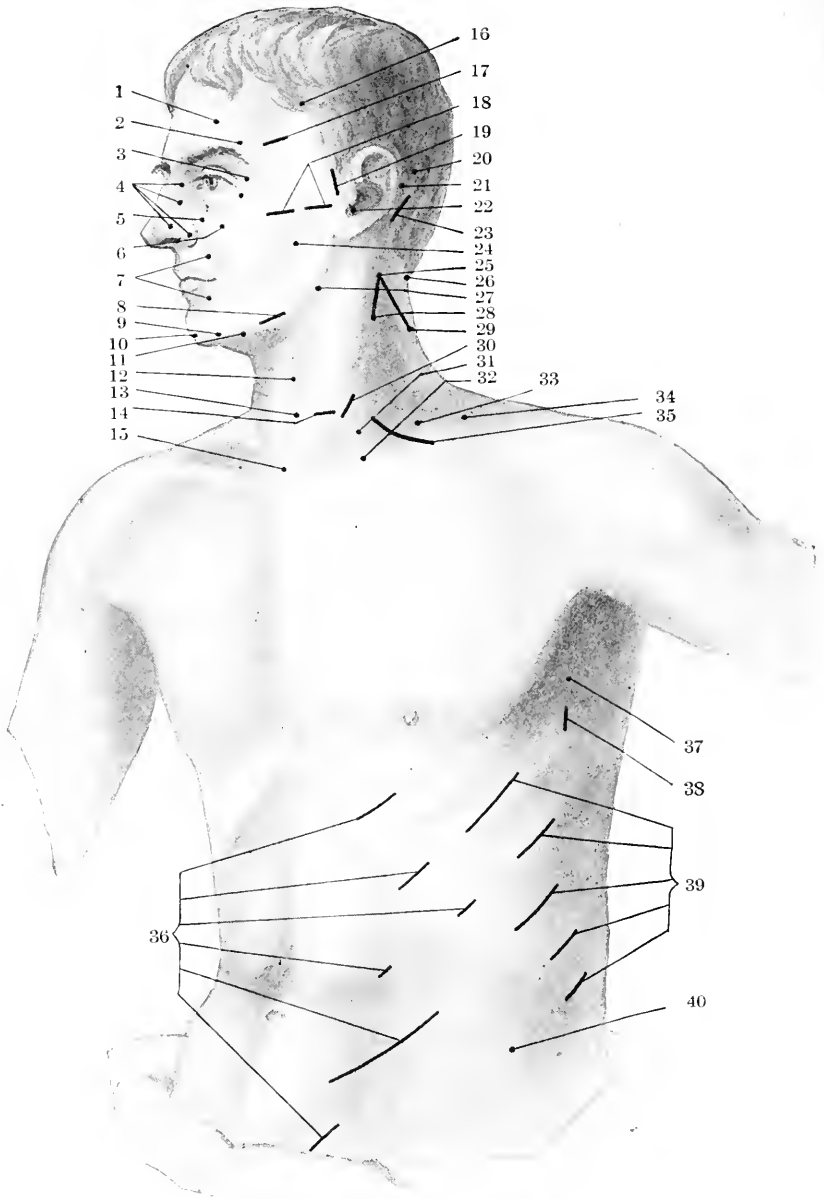


FIG. 141.—Motor points. 1, frontalis; 2, corrugator supercilii; 3, orbicularis palpebrarum; 4, nasal muscles; 5, levator labii superioris; 6, zygomaticus major; 7, orbicularis oris; 8, lower branch of facial; 9, depressor labii inferioris; 10, levator labii inferioris; 11, depressor anguli oris; 12, platysma; 13, sternohyoid; 14, omohyoid; 15, sternothyroid; 16, temporalis; 17, facial nerve, upper branch; 18, facial nerve, middle branch; 19, facial nerve, lower branch; 20, occipitalis; 21, retrahens aurem; 22, facial trunk; 23, posterior auricular nerve; 24, masseter; 25, spinal accessory nerve; 26, splenius; 27, hypoglossal nerve; 28, sternocleidomastoid; 29, trapezius; 30, phrenic nerve; 31, Erb's point (deltoid, biceps, brachialis anticus, supinator longus); 32, anterior thoracic nerve (pectoralis major); 33, circumflex nerve (deltoid); 34, long thoracic nerve (serratus magnus); 35, brachial plexus; 36, rectus abdominis (nervi intercostales abdominales); 37, serratus magnus; 38, latissimus dorsi; 39, obliquus abdominis externus (nervi intercostales abdominales); 40, transversus abdominis.

This diminution of faradic contractility serves as an early index of the extent of paralysis and atrophy which is likely to appear in acute anterior

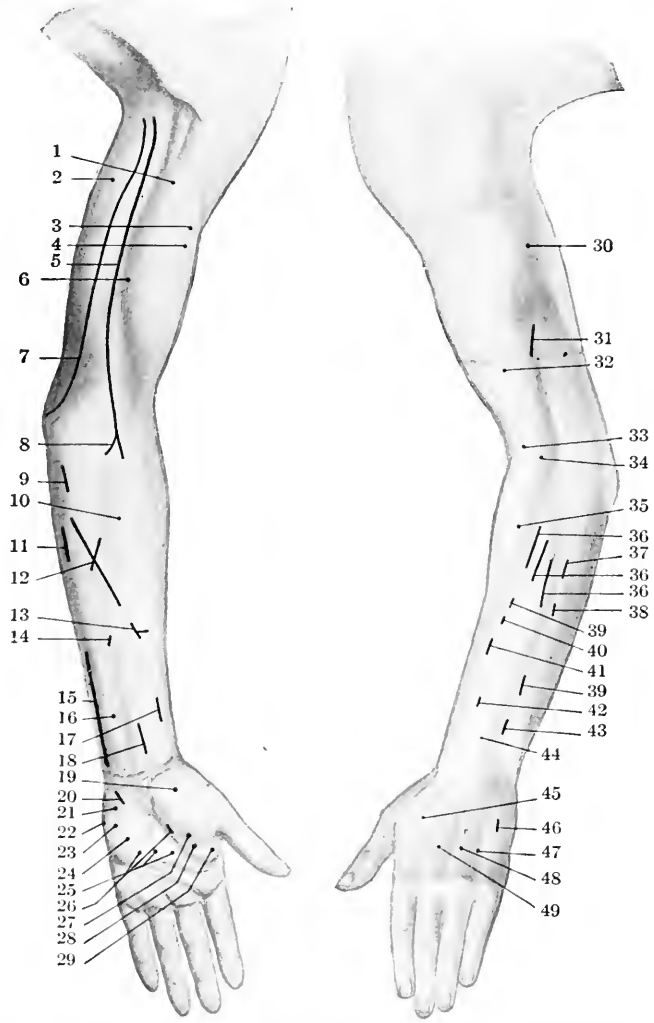


Fig. 142.—Motor points. 1, musculocutaneus; 2, caput internus m. tricipitis; 3, n. musculocutaneus; 4, biceps; 5, medianus; 6, brachialis internus; 7, n. ulnaris; 8, rami n. mediani pro m. pronatore radii terete; 9, palmaris longus; 10, radialis internus; 11, ulnaris internus; 12, flexor digitorum profundus; 13, flexor digitorum sublimis; 14, flexor digitorum sublimis (digitt. II et III); 15, n. ulnaris; 16, flexor digitorum sublimis (digitt. indicis et minim.); 17, flexor pollicis longus; 18, medianus; 19, abductor pollicis brevis; 20, rami volar. prof. nervi ulnaris; 21, palmaris brevis; 22, abductor digiti minimi; 23, flexor digiti minimi; 24, opponens digiti minimi; 25, lumbricales II, III et IV; 26, opponens pollicis; 27, flexor pollicis brevis; 28, adductor pollicis; 29, lumbricalis I; 30, caput externus m. tricipitis; 31, n. radialis; 32, brachialis internus; 33, supinator longus; 34, radialis externus longus; 35, radialis externus brevis; 36, extensor digitorum communis; 37, ulnaris internus; 38, extensor digiti minimi proprius; 39, extensor indicis proprius; 40, extensor indicis prop. et abductor pollicis longus; 41, abductor pollicis longus; 42, extensor pollicis brevis; 43, extensor pollicis longus; 44, flexor pollicis longus; 45, interosseus dorsalis I; 46, abductor digiti minimi; 47, interosseus dorsalis IV; 48, interosseus dorsalis III; 49, interosseus dorsalis II.

poliomyelitis, in Bell's palsy, or other disease inducing rapid degeneration of muscles; but at the end of two weeks from the onset in these affections there is commonly no response whatever to faradism.

On the other hand, if the galvanic current be applied as described above over paralyzed or atrophied muscles the contractility is found to be at first increased; that is, galvanic hyperexcitability is a sign of muscle

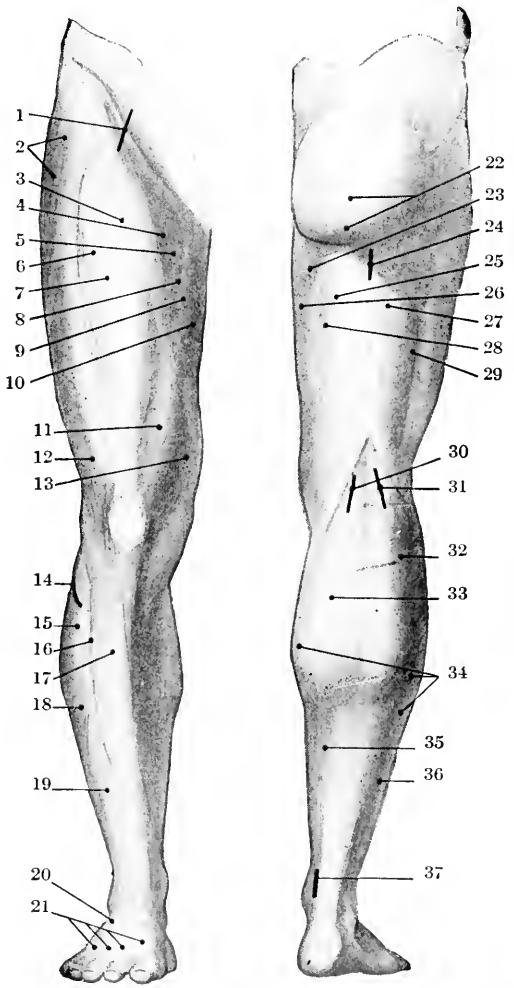


FIG. 143.—Motor points. 1, anterior crural nerve; 2, tensor fasciæ latæ; 3, sartorius; 4, obturator nerve; 5, pectineus; 6, quadriceps (common point); 7, rectus femoris; 8, adductor longus; 9, adductor magnus; 10, gracilis; 11, crureus; 12, vastus externus; 13, vastus internus; 14, external popliteal nerve; 15, peroneus longus; 16, extensor longus digitorum; 17, tibialis anticus; 18, peroneus brevis; 19, extensor hallucis longus; 20, extensor brevis digitorum; 21, dorsal interossei; 22, gluteus maximus; 23, adductor magnus; 24, sciatic nerve; 25, semitendinosus; 26, gracilis; 27, biceps (long head); 28, semimembranosus; 29, biceps (short head); 30, internal popliteal nerve; 31, external popliteal nerve; 32, gastrocnemius (outer head); 33, gastrocnemius (inner head); 34, soleus; 35, flexor longus digitorum; 36, flexor longus hallucis; 37, posterior tibial nerve.

degeneration. Later it diminishes. Contraction of the muscles under the galvanic current is only momentary, appearing both on closing and on opening the circuit. The various responses of the normal muscle are as follows: To the negative pole, or cathode, the first or most active response is on closing the circuit, which is expressed thus, CCC. On opening the

circuit there is no response, C O C. To the positive pole, or anode, a response not so active as to the cathode is obtained on closing the circuit, A C C, and occasionally a response is also obtained on opening the circuit, especially if the pole is held on the trunk of a motor nerve, A O C. These two responses to the anode may be about equal, but usually the response at closure is greater than at opening, and neither is as active as the response to the closure of the cathode. Thus the normal formula stands as follows:

$$C\,C\,C > A\,C\,C > \text{or} = A\,O\,C > C\,O\,C.$$

This formula represents what we find practically at the bedside. There are some distinctions between the responses to nerve-tissue on the one hand and muscle-tissue on the other, as observed in laboratory experiments on animals; but these need not detain and confuse us here.

Reaction of Degeneration—R. D.—When a muscle is degenerating—for instance, when it is cut off from its nerve supply either by injury or disease (nerve injuries, neuritis, acute anterior poliomyelitis)—the reactions to galvanism are altered. The anodal closure contraction becomes greater than the cathodal closure contraction, A C C > C C C, although both are diminished as compared with those of the normal muscle. At the same time the anodal opening contraction (never very conspicuous) disappears, and very rarely the cathodal opening contraction is seen. Thus the typical reaction of degeneration is as follows: A C C > C C C (C O C sometimes seen, A O C disappearing). The response of degenerating muscle is sluggish, not quick and active.

8. Trophic Disturbances.

In a broad sense all disease is nutritional disorder; but there are some diseases which directly attack the nervous structures presiding over nutrition of related parts of the body, and these are properly "**trophic diseases.**" The nutritional disorder may be the principal manifestation of the disease, as is indeed recognized in the very name of the group of muscular atrophies. Whether or not there be separate trophic nerve-fibres, we know that for the muscles the trophic impulses traverse the motor nerves chiefly. If motor palsy is accompanied by rapid wasting, the lesion is probably in the gray matter (of the cord, oblongata, etc.) or in the peripheral nerves, since they, comprising the lower motor neurons, preside over nutrition most directly. But slow wasting may affect parts paralyzed by cerebral disease (upper motor neurons), the affected side in old hemiplegia being commonly much atrophied. This is ascribed to involvement of trophic centres in the cortex. While the spastic spinal palsies arise from disease of superior motor neurons—pyramidal tracts—they often manifest atrophy which may be similar in all respects to that of chronic poliomyelitis—ordinary progressive muscular atrophy. In such cases there is no physiological paradox: the atrophy is referable to implication of the gray matter of the cord. Primary lateral sclerosis is practically always accompanied by atrophy, distributed as in poliomyelitis, which implies that the two motor neurons—superior and inferior—are perhaps independently, though simultaneously, involved, and bulbar palsy is frequently included in the clinical picture. It is well, therefore, to conceive of chronic polio-

myelitis, lateral sclerosis, amyotrophic lateral sclerosis and bulbar palsy as constituting one disease, of which a particular symptom—atrophy, etc.—is dominant in each of the types named.

The distribution of muscular atrophy has considerable significance, especially the region of the body in which it first appears. Atrophy beginning in the small muscles of the hand, or in the shoulder, is generally progressive—spinal—muscular atrophy.

In the "family type" of spinal atrophy appearing in infancy, the muscles of the legs and back are the first to show wasting. The myopathies or muscular dystrophies are likely to appear first in the pelvic girdle (leg type), in the shoulder girdle (arm type), or in the face (face type). When atrophy occurs in the foot and outer lower leg—peroneal muscles—the so-called primary neuritic atrophy is to be considered.

The cardinal tests of spinal, as distinguished from idiomuscular, atrophies are the electrical reaction of degeneration and fibrillary twitching, both present in the former, and absent in the latter or myopathies.

FIG. 144.—Bed-sores.—German Hospital.

The muscles above or below a diseased joint often waste. This is called "arthritic atrophy," and is explained as a reflex phenomenon set up by irritation of sensory nerves supplying the joint.

The clinician must discriminate between the atrophy from disuse or from joint disease, and that which is the essential manifestation of certain grave nervous diseases; and he does so mainly by considering the correlated symptoms and signs.

Certain diseases are presumably, though not manifestly, trophic in origin; as arthritis deformans. Others, such as acromegaly, myxœdema, and adiposis dolorosa, result from disease of ductless glands—pituitary body, thyroid—through the medium of the trophic nervous apparatus, which is affected by the absence or derangement of the secretions of these glands. These diseases—characterized respectively by gross enlargement of hands, feet, and face; by thickened, doughy skin; by great masses of painful fat—illustrate trophic excess, hypertrophy, and hyperplasia.

Still another group of affections, pathologically obscure and clinically indefinite, illustrate trophic disease arising through the medium of vasomotor derangement. These comprise angioneurotic œdema, acroparæsthesia, Raynaud's disease, erythromelalgia, and perhaps other diseases.

A variety of local affections occur however as incidents, more or less important, in the course of organic nervous diseases, and constitute trophic manifestations of these diseases, just as paralysis and anæsthesia constitute

FIG. 145.—Ataxic elbow-joint.—Young.

their motor and sensory manifestations. These trophic disturbances sometimes resemble independent affections and the nervous diseases underlying them may thus be overlooked, for instance, bed-sores in emaciation or sprains in Charcot joints. A joint affection, especially if it be subacute or

FIG. 146.—Perforating ulcers of the foot.—German Hospital.

chronic and confined to one or two joints, is often of nervous origin. In acute myelitis inflammatory arthropathies, resembling rheumatism, may arise. In old hemiplegia—on the paralyzed side—and in various chronic cord-diseases, an osteo-arthritis is not uncommon. The classic form is the Charcot joint, which occurs most frequently early in the course of

tabes dorsalis. The arthropathy of syringomyelia often affects the spine, inducing scoliosis. Painless whitlows of fingers or toes call for study of sensation in these parts, for if they betray loss of temperature and pain sensibility, we are dealing with "Morvan's disease," a trophic manifestation of syringomyelia. Painless perforating ulcer of an extremity, often on the ball of the foot or great toe, belongs commonly to tabes dorsalis or to syringomyelia. Bed-sores form usually at spots injured, especially over the sacrum from pressure of the bed, but the extent of the ulceration is ordinarily out of proportion to the apparent cause. Moreover, sloughs do form without external cause, from purely trophic defect. The skin shows changes in various nervous diseases, as do the nails, hair and other structures histologically allied to the skin. "Glossy skin,"—shiny, thin, dry epidermis on the extremities,—results from neuritis of somewhat long duration. The vesicles of herpes zoster are a trophic manifestation of neuritis, most frequently intercostal.

9. Pain and Temperature.

In ordinary anæsthesia, as that of neuritis or of tabes dorsalis, loss of sensibility to pain, and to heat and cold, is associated with the loss of touch sense. The nerves and dorsal roots, the seat of these diseases, contain the fibres for all forms of sensation.

There is a remarkable condition, however, in which a patient, though feeling himself touched by an object—touch sensation preserved—cannot tell whether it is hot or cold—temperature sense lost—or whether it is sharp or dull—pain sense lost. This separate sensory loss is called *dissociated anæsthesia.* It is in the root-zone that the pain and temperature fibres part company with all others to enter the gray matter and sweep across by way of the commissure to the opposite margin of the cord.

In the neighborhood of the central canal—central gray matter—the pain and temperature fibres from one side decussate with those from the opposite side in a narrow space, and a small lesion at this point, sparing the dorsal columns, may cause dissociate anæsthesia. The lesion that most often occurs here is a peculiar tumor that forms by proliferation of neuroglia just back of the central canal.

When proliferated rapidly neuroglia forms a soft mass. In the brain where its commonest seat is deep in the cerebellum, it meets equal pressure on all sides and so becomes globular—glioma; but in the cord the line of least resistance is up and down, and the gliomatous tissue forms a rod along the centre of the cord. Neuroglia tumors tend to break down centrally. Glioma of the brain is thus commonly cystic, and gliosis of the cord when advanced is characterized by cavity formation within it, by which the cord is finally converted into a tube. From this circumstance the entire disease-process gets its name *syringomyelia.*

Dissociated anæsthesia may result from tumor, hemorrhage in the central gray matter, but it is so early and so constant in gliomatosis that it is commonly spoken of as *syringomyelic* dissociation.

As the neuroglia mass spreads it causes various symptoms, most commonly those of progressive muscular atrophy because the ventral gray

horns are slowly invaded. In chronic poliomyelitis it is usually the hands and arms that are atrophied, the cervical and upper thoracic part of the cord being the commonest seat of the gliosis. The pain and temperature fibres from each root-zone, having reached the opposite margin of the cord, turn upward to form the column of Gowers. This column is bounded in front by the motor root-zone, behind by an imaginary line passing transversely through the central canal. A lesion such as tumor severing the column of Gowers will cause dissociated anæsthesia below it on the opposite side of the body. Lateral trauma of the spine is likely to sever this column, with a similar result; but practically such a trauma always implicates also the crossed pyramidal tract, causing motor paralysis on the side of the lesion. The combination of motor paralysis on one side of the body and sensory paralysis on the opposite side, "Brown-Séquard's paralysis," is pathognomonic of unilateral cord lesion. In Brown-Séquard's paralysis touch sense is usually preserved on both sides of the body, the dorsal columns of the cord escaping.

10. Muscular Sense.

Normal coördination depends upon several factors, any one of which being defective, incoördination or ataxia may result. In walking, under normal circumstances, the sensations imparted by the surface control to some extent the movements, and the absence of this control, as in the anæsthesia of tabes, constitutes an element of ataxia. Subconscious sensations from the joints, muscles, skin, fasciæ, together with appreciations of weight and balance, enter into the special kind of perception designated the muscular sense—"sixth sense" of Sir Charles Bell—and defect of this sense is an important factor in most forms of ataxia. It is suppressed at its very source when the nerve-termini in joints and muscles are implicated in a peripheral neuritis, and this causes so marked an ataxia that such cases have been designated peripheral pseudotabes. A part of the ataxia in such cases of peripheral neuritis may be due to anæsthesia of the skin. The ataxia of true tabes has, to some extent, this same peripheral origin, since neuritis is a part of the disease, but it has a more important spinal origin. In the cord many muscular-sense axons pass up the dorsal columns in company with the touch-sense axons, and here they are implicated in tabetic degeneration. Ataxia, by loss of muscular sense and by anæsthesia combined, is a constant symptom of lesion of the dorsal columns.

Muscular sense is represented in the cerebral cortex posterior to the motor area, being associated with touch sense here as in the cord. These two senses are involved when the hand, unaided, recognizes an object held in it (stereognosis); they are especially combined for this purpose in the superior parietal lobule, and loss of this perceptive power—*astereognosis*—is most commonly due to lesion in that area.

Muscular sense guides the cerebellum in its chief function, the maintenance of equilibrium. Fibres delegated to this function from the root-zone enter the base of the dorsal gray horn and connect with the cell-bodies of Clarke and Stilling which are found in that situation throughout

the cord (Gordinier). These cell-bodies are the beginning of superior muscular-sense neurons; their axons sweep outward to the margin of the cord and turn upward in the direct cerebellar tract, the terminus of which is the middle lobe—vermis—of the cerebellum, which it reaches by way of the inferior cerebellar peduncle—restiform body. Lesion of this neuron-system, in the cord or in the cerebellum (Barker), causes the defect of equilibration called cerebellar ataxia.

Assistance in coördination is derived from all the senses, consciously, as when the tabetic watches the ground in walking, and unconsciously, through impulses collected in the cerebellum from the eye, cutaneous sensations, the joint and muscle surfaces and the internal ear. Disturbance in one of these sensory organs may cause vertigo.

The internal ear is virtually two organs, having distinct functions, and the eighth nerve is double accordingly. The semicircular canals of the vestibule are water-levels telling the position of the head, as muscular sense does that of the limbs, and the part of the eighth nerve arising thence called the vestibular nerve is concerned not with hearing but with equilibration. It connects with its superior neurons in the dorsomesal nucleus to pass to the cerebellum.

Lesion of any part of the vestibular tract from the internal ear to the cerebellum may cause vertigo, as in Ménière's disease.

The eighth nerve's division into two is clear as it enters the pons, the two parts being separated by the inferior cerebellar peduncle. The outer or cochlear division is the true nerve of hearing. It enters the ventrolateral nucleus to be continued by fibres that cross the middle line of the pons, forming the trapezoid body,—acoustic decussation (M. Allen Starr),—then pass upward in the lateral fillet, and by way of the postgeminum and postgeniculum reach the auditory centre in the first temporal convolution.

The Stigmata of Degeneration.

Degeneration, degeneracy, deviation are terms used to denote in individuals a decline from the average normal condition in physical or moral qualities. This decline varies in degree from deviations from the normal scarcely to be recognized upon the most careful study, to the possession of physical and moral defects which render the subject unfit for the ordinary duties and responsibilities of life, and are obvious to the casual observer. It is accompanied by physical, physiological and neuropsychic anomalies known as the "stigmata of degeneration." An undue importance has doubtless been ascribed to these anomalies and their combinations, especially to those of minor degree, by Lombroso and his followers; nevertheless their consideration is of practical value in the study of diseases of the nervous system and has an important bearing upon the diagnosis and prognosis of individual cases of this group of affections. Every sign of deviation from the average normal is not necessarily a stigma of degeneration, and Walton has suggested that it is desirable "to name the phenomena signs of deviation, and call their possessors deviates or a deviate as the case may be, limiting the term degeneration only to such deviations as obviously imply deterioration."

Etiological Classification.—Walton has grouped the causes of the so-called stigmata of degeneration, including the signs of deviation only, as follows:

I. The potential variations from the average normal contained in the parent germ, including the results (a) of atavism, (b) of parental similarity, and (c) of selective tendency on the part of the ancestry.

II. Intra-uterine infection.

III. Mechanical injury during intra-uterine life.

IV. The absence or peculiarity in the germ of certain elements, or their disappearance or anomalous development, without traceable inherited tendency or other known explanation.

V. Mechanical influence exerted during infancy.

VI. Deleterious influences and habits in the ancestry, productive of enfeeblement, undersize, and lessened resistance in the progeny but not altering the essential potential characteristics transmitted by the parent germ.

VII. Absence or hypertrophy of certain glands, pituitary, thyroid, which have a nutritional influence.

VIII. Arrest of development, such as is seen in harelip and similar defects.

List of Stigmata.—The following list, compiled from Dana, Church and Peterson, Walton, and others, includes the more important deviations and stigmata. Those which are of minor significance, either alone or in association with others, as indicating actual degeneracy, are placed in middle single columns; those generally recognized as stigmata of degeneration, in double columns at the sides.

Anatomical Stigmata.

Anomalies of the Cranium.

Cranial asymmetry.		Macrocephalus.
Microcephalus.		Platycephalus.
Leptocephalus.		Oxycephalus.
Plagiocephalus.		Scaphocephalus.
Trigonocephalus.		Short parietal arc.
Short frontal arc.		High prominent forehead.

Anomalies of the Face.

Heavy jaws.		Prognathism.
Lemurian hypophysis.		Opisthognathism or retrognathism.
Orthognathism.		
Large frontal sinuses, small orbit.		Crania progenæa (lower teeth projecting beyond upper, and inferior maxillary angle obtuse).
Great or unequal prominence of malar bones.		

Anomalies of the Eye.

Narrow palpebral fissure.		Microphthalmus.
	Flecks on the iris.	
	Chromatic asymmetry of the iris.	
Albinism.		Congenital cataracts.
Pigmentary retinitis.		Muscular insufficiency, strabismus.
	Hypermetropia.	
Myopia.		Astigmatism.

Deformities of the Palate.

High and narrow.		Horseshoe.
Torus palatinus.		Gothic arch.
Dome-shaped.		Flat-roofed.
Hip-roofed.		Asymmetrical.

Dental Anomalies.

Badly set and badly nourished.		Small or peg-shaped lateral incisors.
	Double rows.	
	Adventitious teeth.	
	Double crown.	
	Macrodontism.	
Microdontism.		Projecting teeth.
Badly placed or misplaced teeth.		Striated transversely.
Hutchinson's teeth.		

Anomalies of the Nose.

	Deviation of nose.	
Defective development of cartilage and tissue of alæ.		Absent cartilages.
		Atresia of nasal fossa.
		Defective osseous development.

Anomalies of the Tongue and Lips.

	Macroglossus.	
	Microglossus.	
	Bifidity of point.	
	Harelip.	
	Cleft palate.	

Anomalies of the Ear.

	Excessively long.	
	Excessively prominent.	
	Set too close to the head.	
Set too far back.		Set too low.
Absence of helix, antihelix, or lobule.		Obliteration of markings.
Absence of fissura intertragica.		Too conchoidal (antihelix, crura, etc., too little marked and helix like rim of funnel).
	Excessively large (absolutely or relatively).	
Too small.		Lack of uniformity in width.
Asymmetry of the two ears, general anomaly of left (Blainville ear).		
	Prominence of antihelix.	
	Adherent lobules.	

Anomalies of the Limbs.

	Left arm and leg longer than right.	
	Excessive length of arms.	
	Long fingers.	
	Polydactyly.	
Symphysodactyly or achistodactylus (joining of fingers).		Syndactyly (web fingers).
Ectrodactyly (fingers wanting).		Amelus or ecromelus (limb wanting).

Anomalies of the Limbs.

Phocomelus (segment of limb wanting).		
	Megalomelus.	
	Megalodactyly.	
Oligomelus (excessive gracility).		Oligodactyly.

Anomalies of the Trunk and General Structure.

Hernias, when congenital.		Malformation of breasts and thorax.
Dwarfism.		Gigantism.
Infantilism.		
Masculinism in women.		Femininism in men.
	Spina bifida.	
Feebleness of construction.		Lordosis.
Scoliosis.		Kyphosis (Fere).
Malformed coccyx.		Thoracic asymmetry.
Mammary development in males.		Mammary absence or reduplication in females (polymastia).

Anomalies of the Genital Organs.

Small or deformed genitalia.		Hermaphrodism.
Cryptorchismus.		Hypospadias.
Epispadias.		Defect, or great volume of prepuce.
Torsion of prepuce.		Imperforate meatus.
Labia too large or too small.		Clitoris large.
Labia minora hypertrophied.		
	Folds between labia majora and minora.	
	Labia minora pigmented, particularly in brunettes.	
Imperforate vulva.		Atresia of vagina.
Double vagina.		Uterus bicornis.
Atrophic uterus.		

Muscular Anomalies.

Dystrophies.		Unequal innervation of facial muscles on the two sides.
Depression above glabella due to overaction of corrugators.		

Anomalies of the Skin.

	Polysarcia.	
	Hypertrichosis (superfluous hair).	
	Premature grayness.	
	Precocious and abnormal hairy development.	
	Rudimentary tail.	
Glabrous chin (no beard).		Vitiligo.
Absence of nails or fetal state of nails.		Melanism of skin.
	Pigmented or vascular nævi.	
Molluscum.		Ichthyosis.
	Pigmented spots.	

Physiological Stigmata.

Anomalies of Motor Function.

Lefthandedness.		Tremors.
Retardation of learning to walk and talk.		Epilepsy.
Nystagmus (congenital).		Tics.—Facial spasm, habit chorea, tic convulsif.

Anomalies of Sensory Function.

Deaf-mutism.		
	Neuralgia.	
	Migraine.	
	Constitutional headaches.	
Hyperæsthesia.		Anæsthesia.
Blindness.		
	Daltonism (color-blindness, achromatopsia).	
	Hemeralopia (night-blindness).	
Nyctalopia (day-blindness).		Concentric limitation of visual field.

Anomalies of Speech.

Mutism.		Defective speech.
	Stammering.	
	Stuttering.	

Anomalies of Genito-urinary Function.

Sexual irritability.		Impotence.
Sterility.		Urinary incontinence.
Amenorrhœa.		

Anomalies of Instinct or Appetite.

Gluttony.		Merycism.
Rumination.		Uncontrollable appetites (narcotics).

Retardation of Puberty.

Deficient Vital Activity of Organic Functions.

	Weak heart.	
	Low arterial tension.	
	Coldness of extremities.	
	Flushing of extremities.	
	General chills and flushes.	
	Weak digestion.	
	Constipation.	

Psychic and Psychoneurotic Stigmata.

Dementia præcox.		Compulsive insanity.
Mania depressive.		Melancholia.
Hysteria.		Hypochondria.
Phobia.		Psychopathic endowment.
Invalid habit.		Idiocy.
Feeble-mindedness.		
	Eccentricity.	
Moral delinquency.		Sexual perversion.
Precocity.		Over-development of certain aptitudes.
Paranoia.		Ideo-obsessive constitution.

X.

THE EXAMINATION OF THE EYE.[1]

General Considerations.

The close relationship existing between the eye and the nervous system, the opportunities furnished by the fundus of the eye to study changes in the *general* circulatory system, and the knowledge that disturbances of ocular function are not infrequently the underlying cause of systemic affections, render a study of the ocular apparatus of extreme importance in the diagnosis of general disease.

It must be remembered that, while the eye is the organ of sight, with its own special function, it is also a part of the general organism, is influenced by the status of the whole body, is disturbed with the disturbance of other structures, and exhibits in a marked degree affections of other organs by which its function is interfered with.

The importance of the thorough analysis of the ocular complications in all diseases, particularly in affections of the brain and spinal cord, is well recognized. An examination of this character, to be effective, must be systematized in order to determine the actual conditions underlying an affection of which the eyes furnish the chief manifestation; in what respect, if any, the ocular functions are abnormal; and finally, the true inference to be drawn from these disturbed functions in the diagnosis of systemic affections.

1. Inspection: The position of the eyeballs in relation to the orbital bones is observed to determine any undue prominence or recession of one or of both eyes; any abnormality of the eyelids as evidenced by tumors, general swelling, drooping, inability to close the lids, inverted margins, size of the commissure, and the presence of crusts or secretions on the margins; congestion of the blood-vessels, or granulations or new growths on the conjunctiva; the size, response to light stimuli, and equality or inequality of the pupils, and variations in the color of the irides; deviation of the visual axes, or involuntary movement of the eyeballs; the sensibility of the cornea or its loss of transparency; and the depth of the anterior chamber and any turbidity of its fluid contents.

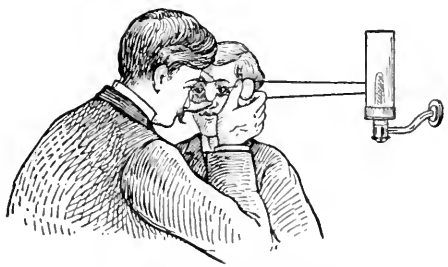


Fig. 147.—Oblique or focal illumination.—From Hansell and Sweet.

The anterior segment of the eyeball is most satisfactorily studied by *oblique illumination*. The patient is placed about two feet from the source of illumination. The examiner focusses the light upon the cornea with a convex lens of 2-inch or 3-inch focus held between the thumb and forefinger of the right hand, and studies the illuminated area through another lens of similar strength held between the thumb and forefinger of the

[1] Contributed by Professor Sweet as collaborator.

left hand, the second finger raises the upper lid, and the little finger resting upon the forehead steadies the hand. The distance of the second lens from the eye is varied slightly to bring into focus the cornea, iris, and crystalline lens. Opacities of the cornea or lens, as seen by oblique illumination, appear as gray or white spots upon the black background of the pupil.

2. Vision: Decrease in the normal acuteness of vision of each eye as measured by test letters for near and far is to be noted; the history of the decline, and its association with pain or inflammation of the external structures; any departure from the normal field of vision must be recorded; contraction of the peripheral limits for form and color, areas of deficient or lost perception, and reversal in the order of the color fields.

3. Ophthalmoscopic Examination: Two methods are employed in the examination of the deeper structures of the eye by the ophthalmoscope—the Direct Method, which gives an upright image of the eyeground, and the Indirect, in which the image is inverted. In both the patient is seated in a darkened room with his back to the source of illumination, and the observer is to the side to be examined. By the direct method the examiner approaches close to the side of the patient's head, using his eye corresponding to the eye under examination, and reflects the light by means of the ophthalmoscopic mirror into the eye. The rays from the fundus are reflected back, and, passing through the opening in the mirror enter the observer's eye, giving an upright image of the eyeground. The optic nerve is best seen when the patient looks at a distant object to the side and beyond the observer's head. The foveal region is brought into view when the patient's gaze is directed into the aperture of the mirror. By the indirect method the observer, about 15 to 20 inches in front and to the side of the patient, reflects the light through a convex lens of about 2-inch focus held at its focal length from the eye, and secures an aërial image focussed by the strong glass. In case the details of the fundus are not at first plainly seen the object lens is slightly advanced or withdrawn from the eye. Strain on the examiner's accommodation is relieved by a +4 D. lens rotated before the sight-hole of the ophthalmoscope.

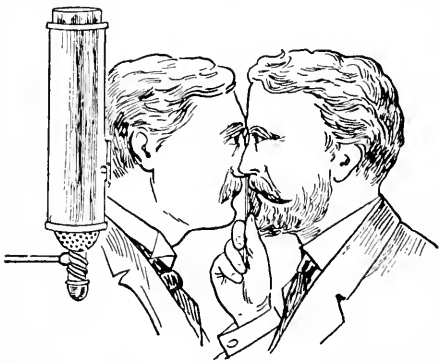
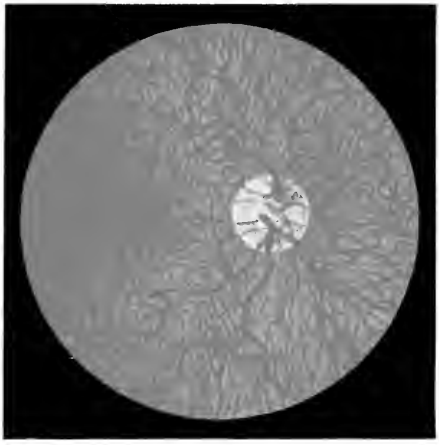


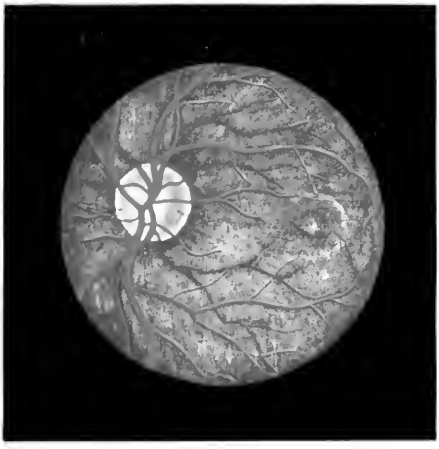
Fig. 148.—Ophthalmoscopic examination by direct method.—From Hansell and Sweet.

The normal eye presents many variations from the typically pictured fundus, and extended experience is necessary to distinguish the variations in health from the changes wrought by disease. The color of the fundus reflex is a bright pink or red, due to the reflected light from the choroidal vessels and the pigment of the retina and choroid. In the negro the reflex is grayish, because of the absorption of the light rays by the abundant pigment. The optic disk, or nerve head, lies to the nasal side of the posterior pole, and is round or oval, with clear cut edges, often fringed with choroidal pigment. The nerve is often cupped in the centre, at which point the central artery and vein



A

B



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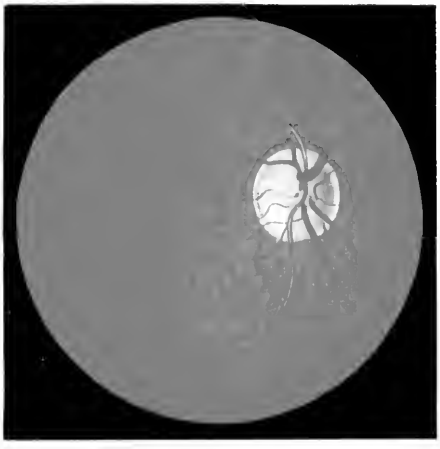

D

[Varieties of the Normal Fundus.—After Würdemann in Posey and Spiller.

A, albinotic fundus; albino and light blonde (after Greef, modified by Würdemann). B, the tessellated fundus; brunette (after Greef, modified by Würdemann). C, the negroid fundus; negro (Würdemann). D, the yellow fundus; Chinese (after Oeller, modified by Würdemann).—D.]

pass. The artery and vein divide into two main branches, and these subdivide into the numerous smaller vessels. The fovea, with its central yellow spot, is the most sensitive part of the retina. It is about 3 mm. to the temporal side of the nerve, and is darker than the rest of the retina. In this region no blood-vessels are to be seen by the ophthalmoscope.

4. Pain: The character of the pain should be known, its situation, its dependence on the use of the eyes, and its association with tenderness in the region of the orbit, particularly at the points of exit of the supra-orbital or infra-orbital nerves.

5. Headache is one of the most prominent symptoms of eyestrain. It is dull and heavy, usually bilateral, increased by application to close work, riding in cars and shopping, and sometimes accompanied by pain in the eyeballs. It is to be distinguished from the sharp periodic attacks of pain characteristic of neuralgia of the first and second divisions of the 5th nerve.

Affections of the nasal tissues, as deflections of the septum and purulent collections in the frontal sinus, cause headache which resembles that of eyestrain. The diagnosis of nasal and sinus headache is made by its longer duration, its association with manifest symptoms of nasal trouble, and its independence of use of the eyes.

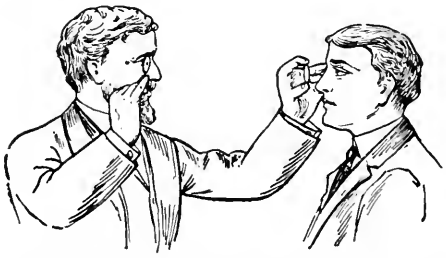

Fig. 149.—Ophthalmoscopic examination by indirect method.—From Hansell and Sweet.

Asthenopia from general muscle weakness is present during convalescence from acute fevers or prolonged illnesses, and attempts at reading are often followed by headache, blurring of sight, and pain in the eyes and head. DeSchweinitz refers to a peculiar form of asthenopia seen after the presbyopic age, most frequently in women, which is not relieved by glasses or treatment of muscular anomalies. These patients present the ordinary symptoms of neurasthenia, doubtless the outcome of beginning arteriosclerosis, and proper tests usually show high arterial tension, which, if reduced by appropriate dietetic and medicinal measures, will cause a disappearance of the asthenopia.

6. Photophobia is a symptom of affections of the cornea and iris, of a few diseases of the retina, and in many cases of uncorrected refractive errors and muscular anomalies. As an isolated symptom it possesses little importance in arriving at a differential diagnosis of ocular affections.

7. Epiphora: Increase in the flow of tears is seen in exophthalmic goitre, in certain affections of the central nervous system (locomotor ataxia), and in obstruction of the lachrymal duct.

The Eyeball and Orbit.

Protrusion of the eyeball—exophthalmos, proptosis—may be caused by tumors, aneurisms, hemorrhage, exostoses, or inflammations originating in or extending to the orbit from the adjacent sinuses; by orbital cellulitis, sinus thrombosis, and paralysis of the ocular muscles.

Bilateral exophthalmos, varying from a slight prominence of the eyeballs to a protrusion that prevents the closure of the eyelids with no inflammatory signs, is found in exophthalmic goitre. Widening of the palpebral fissure from nervous affections, and from the instillation of cocaine, with undue exposure of the sclera, will give the impression of exophthalmos.

Proptosis associated with deep-seated pain upon attempts to move the eyeball, limited or complete immobility of the globe, and swelling and œdema of the eyelids, which may be so great as to prevent opening of the lids, occurs in thrombosis of the cavernous sinus. Inflammations in the orbit, facial erysipelas, with infection carried to the sinus by the facial and ophthalmic veins, infective inflammation of the tonsils, nasal cavities and accessory sinuses may be causative factors. The symptoms are gradual in their development, affecting at first one eye, later the other. Inflammatory exophthalmos is also found in orbital cellulitis due to growths or infection from adjacent sinuses, and rarely after scarlet fever, typhoid fever, and influenza.

Sudden exophthalmos in infants, with the eyeballs turned down, may be due to scurvy. The protrusion may be moderate at first and increase during 24 hours and be associated at its height with thickening and ecchymosis of the upper lid. The subperiosteal hemorrhage to which the affection is due may affect both orbits, but unequally. The eyeball is freely movable.

Pulsating exophthalmos, usually unilateral, most frequently follows traumatism, and is found in arteriovenous aneurism of the internal carotid and cavernous sinus or aneurism of the ophthalmic artery.

Abscess of the frontal sinus may cause displacement of the eyeball downwards and outwards, with diplopia. In purulent disease of the frontal and ethmoidal sinuses a small fluctuating swelling may appear at the upper and inner angle of the orbit, which breaks and discharges pus. Gradual displacement of the eyeball forward may be the result of an orbital tumor within the cone of muscles.

Exophthalmic Goitre.—One of the earlier signs is lagging of the upper lid when the eyes are slowly rotated downwards (Graefe's sign). There is also imperfect power of winking (Stellwag's sign); retraction of the upper lid and widening of the palpebral fissure (Dalrymple's sign), and imperfect power of convergence of the eyes.

Involuntary resistance to eversion of the upper eyelids may be one of the earliest symptoms of Graves's disease. It tends to disappear with the development of the disease, and is explained by hyperexcitability of Müller's muscle through the sympathetic. Swelling of the tissues between the eyebrow and eyelid is an early diagnostic sign of the disease.

Retraction of Eyeball.—Enophthalmus, or sinking of the eye into the orbit, occurs in some instances in extreme emaciation from absorption of orbital fat, in paralysis of the sympathetic, in facial hemiatrophy, and from traumatism. Traumatism in the vicinity of the orbit is sometimes followed by an actual enophthalmus, which may be immediate, or be delayed for several weeks or months.

Nystagmus is a series of involuntary, regular, and rapid oscillations of the eyes. These movements may be horizontal, vertical, or rotary, or

a combination of all three. Unilateral nystagmus is rare. The lateral oscillation is the most common. Congenital nystagmus is found in children with congenital cataract, dense central corneal opacity, or imperfectly developed eyeballs, central choroiditis, and in albinism. Miner's nystagmus is an acquired form, probably due to the prolonged upward inclination of the eyes in semi-darkness. Nystagmus may be a symptom of irritation or diseases of the inner ear, of tumors of the cerebellum, multiple sclerosis, hereditary ataxia, and of syringomyelia.

In multiple sclerosis and in hereditary ataxia nystagmus occurs only when the eyes are turned in the direction of a moving object, and particularly as the eyes reach the limit of their rotation in the lateral plane. A slight nystagmus is occasionally found in hysteria.

Tension.—In the normal eyeball the tension of the globe, as measured by the pressure of the two index fingers upon the sclera through the closed lid, presents a uniform resistance. Increase in the intra-ocular tension occurs in acute glaucoma, in some forms of iridocyclitis, irritation of the cervical sympathetic, intra-ocular tumors, and occasionally after traumatism. Lowered tension may result from degeneration of the ciliary body and choroid, rupture of the globe, detachment of the retina, diabetic coma, and after operations.

The Eyelids.

Marginal Inflammation.—Red and swollen lid margins, associated with heat, burning and photophobia, are seen in persons exposed to cold winds and dust, in children affected with nasopharyngeal inflammation following measles, and as a result of the strain of uncorrected refractive errors. In severe types of the disease the lid margins are covered with crusts which, upon removal, expose ulcers extending deep into the border.

Redness and itching of the lid margins in children may be due to the presence of the pediculus pubis in the eyelashes. Close examination will show the eggs upon the cilia, and the parasite partly buried in the hair follicle.

Inversion of the lashes or of the lid border is most commonly caused by chronic inflammation of the palpebral conjunctiva. The irritation of the cilia leads to inflammation and haziness of the cornea. Eversion of the lid may follow burns or wounds, with subsequent cicatricial contraction of the skin; appears as a senile condition, from loss of muscle power; or accompanies facial palsy. The lower lid is most frequently affected.

Œdema of the lids accompanies severe inflammation of the conjunctiva, purulent disease of the eyeball or orbit, infection of the cavernous, frontal or ethmoidal sinuses, and in general affections like nephritis and gout.

Localized swelling of the eyelids and conjunctiva, with or without vascular changes of the eyeball, is seen in *neurotic œdema*, urticaria, and disease of the antrum, or may be due to errors of diet. The swelling may be sufficient to completely close the eye, and is accompanied by itching and burning. In a few days the parts return to the normal.

A localized painful swelling of the lid and discoloration of the skin may be either a stye (*hordeolum*) or an abscess of the lid (*phlegmon*). The pain is severe and is frequently accompanied by swelling of the entire lid

and œdema of the conjunctiva. Styes are situated in the connective tissue near the lid margin and are apt to recur in persons with deranged bodily functions, and in those who suffer from uncorrected refractive errors.

Swelling of the lid, appearing suddenly after injury, and increasing upon blowing the nose, the soft mass crackling on pressure, is caused by the presence of air which has escaped into the cellular tissue through a fracture of the orbital wall (emphysema).

Inflammation of the tarsus (tarsitis), usually monocular, may be syphilitic, gouty or tubercular. The lid is swollen and drooping and cannot be raised by the levator palpebrarum muscle.

Sebaceous cysts occur both on the eyelids and in the eyebrow. Accumulations of sebum appear as small yellowish elevations which develop about the age of puberty, and are due to improper care of the skin or to gastro-intestinal disorders. Molluscum contagiosum is a disease of the sebaceous glands which occurs among ill-nourished children. It is characterized by waxy-colored, rounded papules, the size of a pea.

Erysipelas attacks the lids by extension from the adjoining skin of the face. The shiny, dusky swelling, with subsequent development of small vesicles, serves to distinguish it from other affections. Severe attacks may affect the orbital tissues and cause optic nerve atrophy and fatal meningitis.

Syphilis.—The primary lesion most frequently occurs at the lid border, near the inner canthus. The ulceration and induration present the typical features of a chancre. It may be mistaken for an epithelioma, but the improvement under antisyphilitic treatment clears up the diagnosis. Tarsitis occurs sometimes in the secondary stage.

Herpes Zoster.—Violent paroxysmal pain, associated with a vesicular eruption on the skin of the lid, forehead and occasionally the side of the nose, is indicative of *herpes zoster ophthalmicus*. The vesicles vary in size, have an inflamed base, and are situated over the region supplied by the first and second division of the fifth nerve. Corneal ulcers, iritis and muscular palsies show ocular involvement. Depressed nutrition is a common cause.

Xanthelasma.—Yellowish irregular shaped patches of connective tissue, with fatty degeneration, located in the upper eyelids near the internal canthus, are termed *xanthelasma*. Rarely they form on the lower lids. The growth is probably due to local ill nutrition.

Chalazion.—A small round elevation of either the upper or lower lid, of slow growth, with the skin freely movable over the hardened tumor, and a purplish discoloration of the surface of the conjunctiva immediately beneath, is termed a *chalazion*. It is due to inflammation of a Meibomian gland, with retention of the secretion. Inflammation of the lid margins and the strain of ametropia may be causative factors.

Inflammation of the Lachrymal Sac.—A small tumor near the inner angle of the lower lid over the lachrymal sac, which disappears upon pressure, with the escape of a viscid mucus through the puncta, is due to catarrhal inflammation of the sac. A similar condition occurs in the new-born from congenital atresia of the lacrimonasal duct. Suppuration with severe pain, and intense swelling and redness of the skin may occur. In both the chronic

and acute forms the tears flow over the cheek. A swelling at the upper and inner angle of orbit, due to sinus disease, may be mistaken for lachrymal abscess.

Ptosis.—Drooping of the eyelid, partially or completely covering the cornea, may be either congenital, or due to injury to the levator muscle, to thickening of the tissues of the lid, to paralysis of the third nerve, or to paralysis of the sympathetic. A form of hysterical ptosis due to spasm of the orbicularis muscle has been described.

Lagophthalmos, or inability to close the eyelid, may be congenital, but is usually an accompaniment of paralysis of the facial nerve. Marked protrusion of the eyeball, mechanically preventing closure of the lid, is seen in orbital tumors, exophthalmic goitre, and in corneal staphyloma.

Blepharospasm, or an involuntary contraction of the eyelids, may vary in degree from a slight twitching of a few of the fibres of the orbicularis to a tonic spasm. In its simplest form it is due to uncorrected refractive errors, inflammation of the lid borders, and corneal and conjunctival irritation. Obstinate cases of cramp of the orbicularis arise from reflex irritation of the fifth nerve, through some remote cause that it is often difficult to determine. It is occasionally a hysterical manifestation.

Conjunctiva and Sclera.

The white of the conjunctiva changes to a dull yellow in so-called biliousness, and to a pronounced brownish-yellow color in icterus. In anæmia, tuberculosis, and nephritis the conjunctiva may become pearly white.

Inflammation.—The normal conjunctiva is coursed by a few small blood-vessels which arise from the deep furrow where the membrane is reflected to the under surface of the lids. Redness is the result of a marked increase in the number of blood-vessels from inflammation of the conjunctiva, or, as this membrane covering the eyeball is transparent, to congestion of the deep sclera beneath.

Hyperæmia of the conjunctiva is seen in measles, scarlet fever, hay fever, influenza, nasal catarrh, the strain of uncorrected refractive errors and from exposure to wind, dust, and bright light and heat. The conjunctiva is often inflamed in facial paralysis, owing to the inability of the lids to protect the globe from external irritants. The presence of a foreign body on the conjunctiva or cornea causes many of the characteristic symptoms of an acute catarrhal conjunctivitis.

The ordinary conjunctival inflammations are unattended with severe pain, but are accompanied with a mucous or mucopurulent discharge. In inflammation of the sclera, the affected area is seen to be beneath the loose conjunctiva, while in disease of the iris, ciliary body or cornea, a ring of fine straight vessels surrounds the corneal border. In these latter affections, pain is often quite severe. Since the conjunctiva may be also inflamed in disease of these deeper structures, a diagnosis cannot be made by the appearance of congestion only.

A type of contagious conjunctivitis (acute contagious conjunctivitis) is due to the Koch-Weeks bacillus or to the pneumococcus. A subacute form of conjunctival inflammation, which may occur in epidemic form, is

caused by the diplobacillus of Morax and Axenfeld. Severe inflammation, with swelling of lids, infiltration of the conjunctiva, and a purulent discharge, occurs from the entrance of infection, usually the gonococci, into the eyes of the child from the birth canal (ophthalmia neonatorum). A similar form of inflammation follows the entrance of gonorrhœal pus into the conjunctival sac of the adult (gonorrhœal conjunctivitis). A purulent conjunctivitis may occur in young girls under 10 years of age who have a vaginal discharge. Usually this discharge contains a few gonococci. In institutions the disease frequently becomes epidemic. A vaginal discharge in children, which does not contain gonococci, may cause a mild conjunctivitis.

Diphtheria of the conjunctiva is rare. A membrane forms on the surface of the conjunctiva, presenting the same characteristics as that found in the throat. A pseudomembranous conjunctivitis may be due to the pneumococcus or to streptococcus infection, and to some of the other micro-organisms found in the ordinary types of inflammation.

Roughness or elevation of the conjunctiva of the lids may indicate trachoma or vernal catarrh. Distended and tortuous vessels in the conjunctiva may be due to constipation, auto-intoxication, chronic alcoholism or lithæmia.

Single or multiple blebs appear on the conjunctiva in badly nourished children, often after measles. Eczema of the nares and disease of the nasopharynx are usually coexistent.

Hemorrhage beneath the conjunctiva appears in injuries of the head, and also in severe compression of the abdomen or thorax. It is not uncommon in whooping-cough, after severe vomiting, and in obstinate constipation, the straining causing a rupture of one of the conjunctival vessels. Spontaneous hemorrhage in the aged, especially if recurrent, should direct attention to the possibility of disease of the blood-vessels and to nephritis.

Uric acid deposits are frequently found in the conjunctiva of the lids of gouty individuals.

Tumors and cysts of various kinds may appear in the conjunctiva of the eyeball. Small, yellowish elevations are found near the cornea, usually at the inner portion, but are of little significance. A fleshy fan-shaped growth is often seen in persons past 40 years whose eyes have been subjected to long exposure to wind, dust or sand. The usual situation is over the internal rectus muscle, the apex often extending upon the cornea. Most of the malignant growths appear at the junction of the sclera and cornea.

Inflammation of the sclera is found in association with the rheumatic and gouty diathesis, tuberculous, intestinal disorders and in syphilis. Tuberculous scleritis is secondary to disease of the anterior uveal tract, and is associated with corneal inflammation. In the superficial form of inflammation (episcleritis) there is usually a circumscribed area of purplish discoloration beneath the conjunctiva, most frequently in the region of the external rectus muscle, and slightly raised above the healthy sclera. In disease of the true sclera (scleritis) the inflammation may affect the entire anterior portion, and extend to the cornea, iris, and ciliary body. Affections of sclera are distinguished from conjunctivitis by the engorgement of the deeper vessels, the purplish color, the severe pain, the absence of discharge, and the frequent relapses.

Cornea.

Keratitis.—The cornea is subject to both ulcerative and non-ulcerative affections.

Ulcerative Keratitis.—Loss of sensibility of the cornea, with subsequent ulceration and destruction, is found in affections of the trunk of the fifth nerve or of its ganglion, or after removal of the latter for trifacial neuralgia. The corneal affection is due to a trophic change in the membrane and to the irritation of foreign substances, which are not recognized by the insensitive cornea.

A severe type of corneal ulceration, which may progress to perforation, is found in association with herpes of the region about the eyes, particularly of the lachrymal branch of the trifacial. The disease is preceded by severe burning and neuralgic pain, in isolated spots, upon which are developed the characteristic vesicles.

Phlyctænular Keratitis.—Small blebs, later forming ulcers, occur on the cornea or at the scleral junction in strumous children, and are associated with inflammatory diseases of the nasal passages, often following the exanthematous fevers. Eczema about the nares is usually coexistent Abscess and ulceration may occur in measles, smallpox, scarlet fever and other infections. Exophthalmic goitre may give rise to extensive ulceration owing to constant exposure of the cornea. Severe ulceration of the centre of the cornea may follow the exhaustion of a prolonged diarrhœa, dysentery, or other debilitating illness in the aged.

Interstitial Keratitis.—Inflammation of the deeper layers of the cornea, without ulceration, is frequently seen in children, between 5 and 15 years of age, who have inherited syphilis, and also in tubercular, scrofulous, and other poorly nourished individuals. In its earliest stage the congestion surrounding the cornea is of the deep vessels, there is dread of light, and close examination shows a fine dot-like infiltration of the interstitial layers of the cornea, which later coalesce into the typical bluish white haziness. The affection is bilateral, although months may elapse before the second eye is affected.

Arcus Senilis.—An arc of fatty degeneration within the cornea, but with a clear, narrow band of corneal tissue separating it from the sclera, is present in the eyes of persons of advanced years, although it is sometimes seen before middle life. It possesses no significance. A senile atrophy of the margin of the cornea has been described in association with arcus senilis.

Partial insensibility of the cornea is seen in exophthalmic goitre, and its presence probably explains a number of the other eye symptoms.

The Iris and Pupil.

Pigmentation.—Slight variation in the pigmentation of the irides is not uncommon in health, but difference in color is rare, except in disease. A yellow green color of one iris, while the other is blue or brown, is an early evidence of inflammation of the iris and ciliary body. It also occurs as a congenital condition, and the eye with the lighter colored iris often later develops cataract. Retained metallic foreign bodies often cause the iris to assume the brownish hue to which the term siderosis is given. Inflammation

of the iris occurs in syphilis, rheumatism, gout, tuberculosis, diabetes, and from injuries, primarily in one eye, or in the fellow eye from sympathy. It is accompanied by irregularity and contraction of the pupil, injection of the pericorneal vessels, and frontal pains, usually worse at night. Swelling in the stroma of the iris is a sign of tertiary syphilis. Sarcoma, as a primary disease, is rare.

The Pupil.—Variations in the size of the pupil occur under the influence of light, and in convergence and accommodation. The average size of the pupils, in diffuse daylight, with the eyes fixed on a distant point, is 4 mm. Careful tests of changes in the pupils are of importance in the diagnosis of general affections, particularly of the nervous system.

The normal reactions are as follows:

1. Direct Reaction.—If one eye is excluded, and the patient directed to fix a distant object, the pupil of the exposed eye, when covered by the hand or card, will dilate. Upon removal of the cover it will contract to its previous size.

2. Indirect Reaction (Consensual Reflex).—If one eye is shaded, the other pupil will dilate equally with the shaded pupil, to again contract when the shade is removed. Normally the two pupils should be of equal size, whether one or both is covered or uncovered.

3. Associated Reaction (Reflex to Accommodation and Convergence).—The patient is directed to look into the distance and then converge the eyes on a point, such as a pencil, held about 5 inches from the eye. The pupils contract under the influence of the convergence and accommodation.

4. Sensory Reaction (Skin Reflex).—Stimulation of the sensory nerves of the skin, by pinching the skin of the neck, or by the passage of a faradic brush along the spine, causes slight dilatation of the pupils.

5. Orbicularis Pupillary Reaction (Lid-closure Reflex).—Contraction of the pupils occurs upon forcible efforts to close the lids.

6. Drug Reaction.—Dilatation of the pupil (mydriasis) follows the instillation of mydriatic drugs, and contraction of the pupil (myosis) the instillation of myotics.

7. Cerebral Cortex Pupillary Reflex.—If a patient seated in a dark room, with the eyes fixed at the black wall, and a light placed to shine laterally into the eyes, is requested to direct his attention to the light, without changing the position of the eyes, the pupils will contract. Since the accommodation remains suspended, and the light entering the eye is unchanged, the contraction of the pupil is in some manner connected with the power of attention; the test should therefore be made in cases of nervous disorder.

Myotic Pupillary Tract.—Stimulation of the centre for the third nerve, by the action of light passing along the optic nerve and optic tracts, causes an impulse to pass to the lenticular ganglion, and thence by the short ciliary nerves to the sphincter of the pupil, which contracts, lessening the size of the pupil.

Mydriatic Pupillary Tract.—The dilator muscle of the iris is innervated by the sympathetic. The impulse passes from the medulla into the cord, thence through the first three dorsal nerves to the superior cervical ganglion, to the plexus around the internal carotid, and through the long

ciliary nerves to the ciliary muscle and iris. Stimulation of the centres of this tract causes dilatation of the pupil.

Abnormal Pupillary Reactions.—Failure of the pupil to react, either wholly or in part, is due to a lesion in the iris, in some part of the third nerve, in the centres of the brain, or in the light-conducting paths. Lesions in the iris may be swelling or atrophy, or old or recent attachments from inflammation. Immobility to light stimulus, with preservation of the reflex to accommodation, is one of the important abnormal pupillary changes.

Reflex Immobile Pupil (Argyll-Robertson Pupil).—Loss of reaction of the pupil to direct light, with preservation of the contraction of the iris in accommodation and convergence, comprises the well-known Argyll-Robertson pupil, and is an early symptom of tabes. Although of great diagnostic value when present,—and in the majority of cases it exists in the incipient stages of the disease,—there are rare instances in which it has not been found, even when all other symptoms of the disease have existed for years. Associated with lost light reflex is frequently noticed alteration in the shape of the pupil. The pupil may be of normal size, but more often myosis is found, from implication of the cervical portions of the cord controlling the dilating centres. The Argyll-Robertson pupillary phenomenon is also seen in paretic dementia. The loss of the light reflex in aortic disease is due to the general syphilitic infection.

Dilatation of the Pupil.—The pupil is dilated in glaucoma, in optic atrophy, in diseases of the orbit, in irritation of the cervical sympathetic, in acute mania, in cerebral softening, in extensive disease or injury of the cerebral centres, in complete paralysis of the third nerve, in paralysis of the sphincter of the iris by a blow upon the eyeball, in strong emotion, and when mydriatics have been used. In neurasthenia and hysteria, mydriasis is often present.

Dilatation of the pupil may be caused by an irritation of the dilator pupillary centre or tract (irritative mydriasis), or by a paralysis of the pupil-contracting centre or fibres (paralytic mydriasis).

Unilateral mydriasis, in which the pupil fails to react to direct light but contracts consensually with its fellow, is seen in complete optic atrophy, in which the conductivity of the one optic nerve is lost. The failure of one pupil to retract to separate stimulation of either eye, but contracting upon convergence, while the other pupil reacts to light stimulus of either eye, is seen in tabes and in syphilis. Sudden unilateral mydriasis in which the instillation of a drug can be excluded is a possible early symptom of latent sclerosis of the cord.

Corte claims that in any serious diphtheritic attack failure of the pupils to react to light indicates a fatal termination.

Complete blindness will cause bilateral mydriasis with failure of the pupils to react to light stimulus. A slight contraction of the pupils has been observed in the blind, who are entirely devoid of light perception, after the eyes have been exposed to bright daylight for several minutes.

In mydriasis from drugs, the accommodation is temporarily suspended.

Contraction of the Pupil.—Abnormal contraction of the pupils is due either to irritation of the pupil-contracting centre or fibres, or to paralysis

of the sympathetic. In disease of the central nervous system, the myosis may be due to irritation of the sphincter nucleus; but should mydriasis follow the myosis, it is an indication of the spread of the affection and destruction of the sphincter centre. In irritative myosis the pupil rarely dilates under cover or in a bright light, but acts normally when a mydriatic or myotic drug is instilled. In the paralytic myosis the reaction to light and in convergence is preserved, but the pupils dilate imperfectly when shaded. Mydriatics act imperfectly, but the pupils contract further to myotics. In old age the pupils are usually smaller than in middle life, although perfectly normal in reaction. Inflammations of the iris are always associated with small pupils, and the iris is likely to become attached to the lens capsule. Myosis is seen in the early stages of inflammation of the brain and meninges, apoplexy, abscess, and in other affections which indicate irritation of the part; also in hysteria, toxæmia, and in epilepsy. Paralytic myosis occurs in tabes, general paralysis, spinal meningitis, and destructive lesions of the cord.

Unequal pupils (anisocoria) may point to purely functional affections, such as hysteria and the psychoses, or to grave organic disease, as paresis, tabes, etc. The pupillary phenomena must be studied in connection with other symptoms to arrive at a correct diagnosis. Inequality of the pupils, although the reaction to light remains, is present in many cases of exophthalmic goitre. Bichelonne believes that unilateral mydriasis is an important sign in the early diagnosis of pulmonary tuberculosis.

Alternating mydriasis, in which the dilatation changes from one eye to the other, is occasionally present in general paralysis and in tabes, and has been described as a premonitory symptom of insanity.

Hippus.—An alternate contraction and dilatation of the pupil, occurring under a uniform stimulus of light, is a normal phenomenon, but may be excessive in hysteria, epilepsy, advanced paralysis, early stages of meningitis and mania, and in phthisis.

Hemiopic Pupillary Inaction.—The Wernicke pupil is described under hemianopsia.

Iritis.—Inflammation of the iris may accompany disease of or traumatism to other ocular structures, or be due to constitutional disorders. The principal signs are changes in the color of the iris, injection of the pericorneal vessels, myosis, deposits on the posterior surface of the cornea, and attachments of the iris to the lens capsule. The symptoms are severe brow pain, worse at night, and slowly failing vision.

Syphilis is the most common cause of iritis. The inflammation affects both eyes, one later than the other, usually appears from the second to the eighth month following the initial infection, and is plastic in character. In some instances one or more yellowish brown nodules may appear at or near the pupillary border, resembling the solitary gumma of the tertiary period of syphilis.

It is probable that the iritis, so long considered as due to rheumatism, is septic or toxic in origin, often from the gastro-intestinal tract. The inflammation is usually unilateral, although the second eye may later become affected. In chronic rheumatic subjects the iritis is of a severe and destructive type. The attacks usually recur during a relapse of the rheuma-

tism, or they may be the only evidence of the toxæmia. The so-called idiopathic iritis, in which syphilis, gonorrhœa, and traumatism can be positively excluded, is often secondary to disorders of nutrition or constitutional affections. In these cases the pain is usually of greater severity, the disease more slowly amenable to treatment, and the relapses frequent.

In severe inflammations of the iris, the ciliary body is involved, and the disease is referred to as irido-cyclitis. Uveitis, or inflammation of the iris, ciliary body, and choroid, occurs in rheumatism and gout, diabetes, influenza, anæmia, syphilis, tuberculosis and the specific fevers; also in affections of the teeth, tonsils, accessory sinuses, the prostate and the skin. The disease, which is probably the manifestation of some toxic process, is characterized by moderately deep anterior chamber, hazy cornea and aqueous, pupil not contracted, occasionally a slight increase in the tension of the eyeball, and the deposit in triangular form of small dots on the posterior surface of the cornea.

Irido-cyclitis is often seen during the late stages of gonorrhœa, and recurs with relapses of the disease. The iritis may occur a considerable time after the acute attack, sometimes preceding or following an affection of the joints.

Tubercle of the iris is seen as two or more reddish nodules in the anterior surface or deep in the iris tissue, occasionally in confluent form, in young persons, usually between the tenth and twentieth year. The patients may not present other active manifestations of the tuberculous process. Koch's old tuberculin may be employed for diagnostic purposes.

Ocular Muscles.

Mobility of the Eyes.—Under normal conditions, the eyeballs move in perfect accord in all directions, with no manifest lagging movement in either eye in any of the several rotations. In equilibrium of the ocular muscles, every movement of one eye is accompanied by simultaneous and equal movement of the other, the image of the object upon which the eyes are fixed is formed on the fovea of each eye, and the effort required on the part of any one muscle or group of muscles in sustaining binocular single vision is equal in the two eyes.

Disturbance of equilibrium may be arranged in two groups:

1. Organic anomalies, in which there is double vision in attempts to rotate the eyes in the direction of the affected or paralyzed muscle or group of muscles.

2. Functional anomalies, in which there are:

(a) An *actual* deviation of the visual line of one eye from that of the other, persisting in all movements of the two eyes.

(b) A *tendency* to deviation, which is overcome by increased or decreased innervation to the muscle or group of muscles affected.

Organic Anomalies (Ocular Palsies).—Since binocular single vision can only be maintained if the image of the object falls upon the macula of each eye or upon corresponding points of each retina, any disturbance of the motor apparatus by palsy of one or more of the ocular muscles results in an impression of the object upon non-corresponding points of each retina.

Two images are, therefore, transmitted to the brain, and double vision, or diplopia, results. The symptoms of ocular palsies are: (1) diplopia; (2) limitation of movement of one or both eyes in the direction of the paralyzed muscle; (3) actual deviation; (4) false protection; (5) vertigo; and (6) abnormal position of the head.

Diagnosis of Ocular Palsies.—Paralysis of an ocular muscle is to be suspected if the patient complains of seeing double or tilts the head to prevent diplopia, and complains of vertigo in attempts to fix an object in that portion of the field in which double vision exists. If the paralysis is complete, the eye with the affected muscle fails to rotate past the median line when the object fixed passes to the side to which the affected muscle ordinarily rotates the eye, and in fixation with the affected eye, the deviation of the sound eye (*secondary deviation*) is greater than is the deviation of the squinting eye when the sound eye fixes (*primary deviation*).

Diplopia.—In partial paralysis the limitation of rotation may be so slight as to escape observation. It becomes manifest, however, even in slight degrees, upon the tests for diplopia. The patient, seated in a darkened room, with the head fixed in one position, is directed to follow with the eyes a lighted candle held at a distance of about 10 feet, and moved in all portions within the field of vision. If a piece of colored glass is held before one eye, the images of the two eyes are in this way differentiated. By this test the behavior of the two images in their relative height and distance from each other, and their separation and approximation, as the light is carried up and down, to the right and to the left of the patient, determines which of the muscles is palsied. Special skill and training are essential in the diagnosis of the more complex forms of palsies, and it is unnecessary in this connection to enter fully into details, but the following points will serve to indicate roughly the character of the affection:

1. Double images are seen only when the eyes are turned in the direction in which the paralyzed muscle or muscles normally rotate the eye; in all other directions there is single vision.

2. The image of the eye with the paralyzed muscle (false image) separates from the image of the sound eye (true image) as the object is carried into the field governed by the muscle affected; that is, the distance between the double images increases as the object fixed upon is moved in the direction toward which the paralyzed muscle should rotate the eye.

If the false image is on the same side as the affected eye the diplopia is homonymous; if the false image is projected to the side of the sound eye the diplopia is crossed, or heteronymous.

Homonymous diplopia, with images in the same horizontal plane, indicates paralysis of an external rectus, right externus if the images separate as the object fixed is carried to the right, and left externus if they separate as the object fixed is carried to the left.

Crossed diplopia in the horizontal plane indicates paralysis of an internus, right internus if the double images separate in looking to the left, and the left internus if they separate in looking to the right.

Vertical diplopia in upper field (that is, one image higher than the other) indicates a paralysis of the superior rectus or inferior oblique: if diplopia increases in looking up and to the right, and image of right eye is

higher, paralysis of right superior rectus; if lower, left inferior oblique. Increase in diplopia in looking up and to the left, with image of right eye higher, paralysis of right inferior oblique; if lower, left superior rectus.

Vertical diplopia in lower field shows a paralysis of the inferior rectus or superior oblique. Increase in the diplopia in looking down and to the right, with image of right eye lower, indicates paralysis of right inferior rectus; if higher, left superior oblique. Diplopia increasing down and to the left, with image of right eye higher, shows paralysis of right superior oblique, if lower, left inferior rectus.

Special Palsies. — Paralysis of the Sixth Nerve.—The long course of the sixth nerve at the base of the brain renders it particularly liable to pressure from inflammatory exudation, hemorrhage, and fracture. It is the most frequent of the ocular palsies, and it is indicated by convergence of the affected eye, homonymous diplopia, and inability of the eye to rotate outwards past the median line.

Paralysis of the third nerve is shown by ptosis, the pupil moderately dilated and unresponsive, the power of accommodation abolished, and crossed diplopia, with the eyeball turned outward and slightly downward from the action of the external rectus and superior oblique. In cycloplegia only that portion of the nerve controlling the ciliary muscle is affected. There may or may not be associated paralysis of the sphincter of the pupil (iridoplegia).

Paralysis of the fourth nerve, which controls the superior oblique, is less frequent. There is vertical diplopia in the lower field, the image of the affected eye is the lower, and the distance between the images increases as the eye is rotated downwards and inwards.

Ophthalmoplegia externa is the term employed to designate paralysis of all the external ocular muscles. The affected eye is incapable of movement, and the lid droops and cannot be voluntarily raised. Paralysis of the iris and ciliary muscle is known as *ophthalmoplegia interna*.

Conjugate Palsy.—In this rare affection the individual muscles of each eye possess their normal power to turn the globe in any desired position, but there is inability to rotate the two eyes in associated action. It may affect convergence, so that the eyeballs cannot be converged, although individually capable of internal rotation: or it is shown in loss of associated lateral or vertical movements. In all cases the lesion is central, and involves the centres for conjugate movement, although spasmodic conjugate deviation is seen in hysteria.

Causes of Ocular Palsies. — Paralysis of the ocular muscles may be due to an intracranial or an orbital lesion or to peripheral causes; it may follow meningitis, tumors, hemorrhage, gumma, or vascular changes in the brain; orbital cellulitis, traumatism, and inflammation of the nerve in the muscle. The constitutional causes are syphilis, tuberculosis, diabetes, nephritis, influenza, tabes, rheumatism, diphtheria, and general paresis.

At least one-half of the ocular palsies are considered to be due directly to syphilitic gummatous deposits, syphilitic periostitis in the orbit or along the base, or to degeneration in or close to the nuclei of the nerves. These are exclusive of the indirect syphilitic affections, as manifested in tabes, general paresis, and diseases of the blood-vessels. Nuclear and peripheral

palsies may be caused by rheumatism, diabetes, tonsillitis, influenza, ptomaine poisoning, and by lead, alcohol, tobacco, and other toxic agents. In that variety of ptomaine poisoning known as botulismus, nuclear palsies are frequent. Basal palsies are seen in hemorrhage, meningitis, especially tubercular, abscess, and cavernous sinus disease. The paralyses associated with diabetes mellitus develop suddenly, but usually are of short duration and mostly affect the sixth nerve. Neuralgia of the region about the eye is often associated with the paralysis, and pain in this situation should direct attention to a possible disturbance of the motor apparatus on the same side.

Ophthalmoplegia interna, or paralysis of the ciliary muscle and the sphincter of the pupil, is more frequently unilateral than bilateral, and is seen in syphilis, tabes, and intracranial disease. Either the sphincter or the ciliary muscle may be first affected, and later the external ocular muscles become implicated. It is also found after diphtheria. The lesion is probably nuclear.

Paralysis of the accommodation, destroying the power of reading, is seen in about 5 per cent. of cases of diphtheria, usually affects both eyes, and only rarely is associated with palsy of the iris. Occasionally paralysis of the external rectus is associated with the loss of accommodation. Similar palsies are seen in severe cases of influenza.

Intermittent palsy of one or more muscles is an early symptom of tabes. One eye is generally affected, and the paralysis disappears in a few weeks to again recur. The external rectus is probably the most frequently involved, and next the muscles supplied by the third nerve, either as a group or individually, while the parts supplied by the fourth nerve are rarely affected.

Palsies of some of the ocular muscles, most frequently those supplied by the third nerve, are present in "ophthalmoplegic migraine" and follow the subsidence of pain. The attacks are usually recurrent, the palsy occurring on the same side as the pain. The disease is rare, and should be differentiated from brain tumor.

Functional Anomalies.—Both of the functional defects, the *tendency* to deviation (heterophoria) and the *actual* turning of one visual line from that of its fellow (heterotropia, or functional squint), are due in many instances to errors of refraction, and to disturbance of the relation between convergence and accommodation. There is no paralysis and no double vision.

Latent Deviations (Heterophoria, Insufficiency of the Ocular Muscles).—If there is a lack of equilibrium in the action of the muscles of the two eyes in binocular vision, so that fixation of the eyes is only maintained through an excessive amount of nerve force expended in helping the weak muscle or set of muscles, there follows a train of symptoms which is usually included under the term *muscular asthenopia*. There is more or less constant dull headache, which may be general or localized in the frontal or occipital region, blurred vision, inability to use the eyes at near work, and photophobia. Sometimes there may be vertigo and nausea, confusion of ideas, insomnia, and a feeling of physical exhaustion while in a moving crowd, in attendance at the theatre, or after riding in the cars. Heterophoria is a most active causative factor in many of the reflex nervous disorders. Relief in many cases has undoubtedly followed the correction of

the defects, but does not justify the extravagant claims made that epilepsy, chorea, melancholia, dyspepsia, and other affections are not only primarily due to heterophoria, but are cured after correction of the muscle anomaly.

Forms of Deviation.—The tendency of the visual lines to deviate from the normal parallelism is divided into *esophoria,* a tendency of the visual lines to turn inward; *exophoria,* a tendency of the visual lines to turn outward; and *hyperphoria,* a tendency of one visual line to deviate above that of its fellow. The inward tendency of the visual lines is of relatively less importance as a cause of reflex symptoms than is hyperphoria or exophoria.

To determine the existence of the muscle anomaly, the latent defect is made manifest by means of a prism of sufficient strength to cause diplopia, or by the use of a piece of cobalt glass or a rod of glass held before the eye. The line of light made by the rod is so dissimilar from the image of the other eye that the fusion impulse is abolished, and the eyes take the position of greatest rest. The prism that fuses the double images made by the prism or brings the line of light into the flame seen by the other eye is the measure of the defect. Correction of the refraction is essential to a cure.

Manifest Deviations (Concomitant Squint, Heterotropia).—In this affection there is an actual deviation of one visual line from that of the other, but the squinting eye is able to follow the movements of the fixing eye in all directions; there is no acknowledged diplopia, and the deviation is transferred from one eye to the other, and remains of the same degree upon alternately covering one eye and then the other. The absence of double vision, and the fact that the power of rotation of the eye is not limited, serve to distinguish the functional from the paralytic squint.

Functional squint may be either convergent, divergent, or vertical. The three principal causes of the strabismus are a disturbance in the normal relation between convergence and accommodation, brought into existence by errors of refraction; a weakness of opposing muscles, either through structural changes or disturbed innervation; and unequal vision of the two eyes, so that the normal desire for fusion is abolished. The strabismus may be monolateral, when one eye always fixes and the other always squints; or alternating, when either eye may be used for fixation, since the visual acuity is about the same in each. Squint is an affection of early childhood, often disappearing if proper treatment is instituted at this time.

Vision.

AFFECTIONS OF VISION.

Imperfect vision is due to errors of refraction; to opacities of the cornea, crystalline lens, or vitreous; to disease of the retina, choroid, optic nerve, or central nervous system; or to functional neuroses.

Central vision is tested by means of letters corresponding in size to a fixed standard. The patient, seated 20 feet from the test card, and one eye covered, is asked to read the smallest line of letters that can be deciphered. If the vision thus estimated does not conform to the standard, the various errors of refraction should be excluded before concluding that the reduced vision is the result of disease. This may be done by placing before the eye

an opaque disk with a small central opening. If no organic disease exists vision will conform to the normal. The effect of faulty vision upon the health of patients is oftentimes overlooked. In a person given to any manner of indoor vocation, whose nervous system is at all delicately balanced, an uncorrected eye-strain may give rise to headache, drowsiness, transient vertigo, and sometimes to nausea, irritability of temper, and insomnia. These symptoms are probably more often found when vision is in excess of the normal standard, hence the state of the refraction must be learned in order to determine the extent to which the accommodative strain is responsible for the reflex manifestation.

Peripheral Vision.—In testing the perception of the outlying portions of the visual field, the examination is made of each eye separately, the oculist employing an instrument known as a perimeter, which consists of an arc of a circle, of about 12 inches radius. The eye to be examined is at the

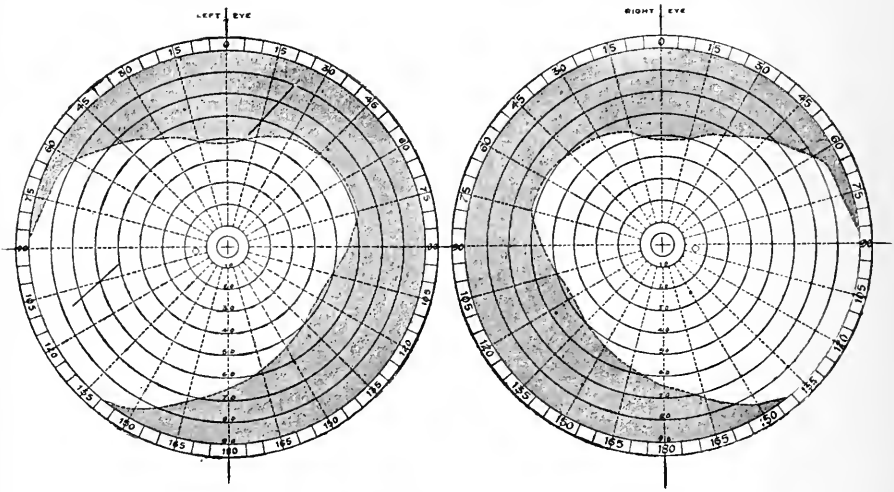

FIG. 150.—Diagram of perimetric charts of visual fields for white (form field).

centre of the circle, and fixed steadfastly upon a white spot upon the arc. A white object 5 to 10 mm. in size is slowly moved along the arc, from its extremity towards the fixed spot, until it comes within the patient's range of vision, and the point recorded at which the object is first seen. The arc is moved to another position, and this is continued until the whole circle has been tested. The record of the usual points so taken is recorded, as in Fig. 150. As will be seen, the outlines of the visual field are far from symmetrical. Its greatest extent is on the temporal side, usually about 90°, on the nasal side 55°, above 50°, below 65°. The perimeter is not absolutely necessary to make out gross lesions such as hemianopsia or extensive contraction of the field, since the finger carried from point to point, as the patient gazes into the examiner's eye, will indicate marked departure from the normal limits. Accurate examination requires the services of the ophthalmologist.

Gradual failure of vision apart from refractive errors is seen in disease of the cornea, in cataract, non-inflammatory glaucoma, atrophy of the optic nerve, and various forms of intra-ocular disease. Rapid loss of sight occurs

in acute glaucoma, retinal hemorrhages, embolism or thrombus of the central retinal vessels, œdema of the retina, cerebral effusions, metastatic disease of the eye, ptomaine poisoning, and after quinine, wood alcohol, and other toxic agents. In every instance of decrease in the normal acuity of vision, the oculist should be immediately consulted.

Cataract affects vision in proportion to the degree and situation of the opacity. It appears as a congenital or senile condition, in connection with disease of the eyes, in diabetes, in traumatism, and with many constitutional disorders that influence the nourishment of the lens through the nutrient vessels of the choroid and ciliary body. Cataract has been mistaken for non-inflammatory glaucoma, owing to the greenish reflex of the lens in the latter disease. The diagnosis is readily made with the ophthalmoscope.

Second Sight.—The ability of persons past middle life to lay aside their usual convex reading glasses and read the finest print (so-called second sight) indicates swelling of the lens, and is one of the first signs of cataract. Glycosuria is a frequent cause of cataract, and acquired myopia after 40 years of age, even with clear crystalline lens, should direct attention to the possible existence of diabetes.

Acute Glaucoma.—Recurring attacks of blurred vision, the obscuration lasting from a few minutes to an hour or more, when associated with halos about the light (iridescent vision), should direct attention in persons past middle life to the possibility of an oncoming attack of acute glaucoma. The "glaucomatous attack" usually occurs at night, is characterized by severe pain in the head, nausea and vomiting, and rapid loss of sight. The eyeball is intensely congested, the pupil dilated, the cornea anæsthetic and steamy, and the globe of stony hardness. The affection should not be mistaken for a "cold in the eye," iritis, or neuralgia. The rheumatic and gouty diathesis is a possible causative factor.

Alterations in the Visual Field.—Changes in the visual field, as evidenced by irregular or concentric narrowing of the normal limits for form and color, the presence of central or peripheral areas of lost perception (*scotoma*), or transpositions of the order of colors, is seen in disease of the retina, optic nerve, and central nervous system, or may be present in purely functional neuroses.

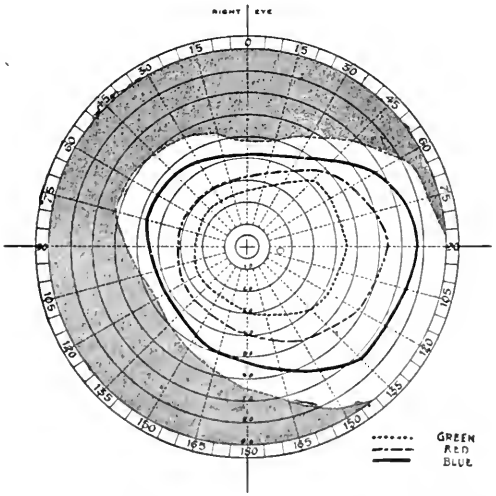

Fig. 151.—Diagram of form and color fields of right eye.

Amblyopia and **amaurosis** designate defective vision due either to functional disturbance or to actual disease of the visual apparatus, without gross ophthalmoscopic changes, although the latter restriction is not always adhered to. The affection of the sight may be limited to central vision, include the whole

or only part of the visual field, or be only for form or for color. A number of congenital forms of amblyopia are recognized—for form, as in the poor vision of squint, or for color, as in color-blindness. Partial or complete loss of sight may be due to irritations affecting the fifth nerve, severe injuries of the head, auto-intoxication, the nephritis of the eruptive fevers, diabetes, malaria, rheumatism, action of certain drugs, and to hysterical manifestations.

Sudden transient failure of vision may mean merely the temporary giving out of eyes already weakened by general affections or too persistent use. In the so-called "visual aura" of migraine, there is a decided blurring of the visual field, which has been designated as amblyopia, but is transitory, and is to be distinguished from the permanent functional impairment of sight included in the term. Dercum regards a slight degree of amblyopia, with or without a diminution of the color sense, as an early and invaluable symptom of paresis which may even antedate distinct and demonstrable anomalies of the pupils or changes in the eye-grounds. Transient blindness, persisting for a few minutes to several hours or days, may be due to spasm of the retinal arteries. The diminution in the calibre of the vessels has been observed in epilepsy, migraine, cold stage of malarial fever, and in some toxic conditions. In uræmia, particularly in the nephritis of scarlet fever and of pregnancy, the sudden loss of sight may be associated with convulsions, coma, and other cerebral symptoms. Although the blindness may be complete, the reactions of the pupils are usually preserved.

Amblyopia from Loss of Blood.—Amblyopia, with subsequent complete atrophy of the optic nerve, may follow profuse spontaneous hemorrhages from the stomach, intestines, uterus, or nasal cavity. The loss of sight may not appear for a week or more after the bleeding, being due, as shown by Holden, to degeneration of the ganglionic cells of the retina from impaired nutrition.

Methyl-alcohol Amblyopia.—Rapid loss of sight may follow the drinking of wood alcohol in its crude or purified state, or when employed as an adulterant in the manufacture of Jamaica ginger, impure whiskey, cheap essences, bay rum, and other alcoholic beverages. The eye symptoms are often associated with vomiting and purging, severe headache, and intense weakness. The vision may improve for a few hours or days to again relapse, often ending in complete blindness. The symptoms of poisoning may follow inhalation of the alcohol by workmen in mixing varnishes, shellacking beer vats, or even when the alcohol is applied locally.

Quinine Amblyopia.—Quinine affects both the optic nerve and retina. Usually the symptoms follow a large dose of the drug, but a moderate amount has been followed by transient visual disturbances. Following a toxic dose there is usually complete blindness, which continues for a few hours, days or weeks. Central vision at first returns, with gradual enlargement of the peripheral field, but the latter may remain permanently contracted. The ophthalmoscope shows cloudiness of the retina, contraction of the vessels, and pallor of the optic nerve. Ethylhydrocuprein, a quinine derivative, employed in the treatment of pneumonia, has been followed by symptoms of quinine poisoning. Felix mas and salicylic acid have caused visual disturbances similar to those of quinine. Nitrobenzole, anilin, iodoform, and thyroidine

effect the visual apparatus when taken in large doses. To this list may be added lead, tobacco, atoxyl, iodoform, osmic acid, chloral and many other drugs used in medicine.

Central Amblyopia (Retrobulbar Neuritis).—The orbital portion of the optic nerve is subject to interstitial inflammation in either an acute or chronic form. In both, the disease affects those portions of the nerve that supply the macular region. The early symptoms are dimness of vision, without marked ophthalmoscopic changes, and a weakness or loss of color perception in the central visual field.

In retrobulbar inflammations the visual acuity is less in very bright light, and exposure to excessive light may lead to deterioration of vision that may last for some time. This is due to the slowness with which the ill-nourished axis-cylinders are regenerated. There is also a close relationship between retrobulbar disease and affections of the seventh nerve, since paralysis of the facial nerve may precede the optic-nerve inflammation. In *acute retrobulbar neuritis* there is rapid failure of vision with central or paracentral scotoma, which is usually followed by recovery of vision, although the optic disk still shows pallor. The affection may, however, rapidly progress until the entire nerve is implicated, and vision is nearly if not completely lost. The disease may arise during the course of rheumatism, gout, diabetes, smallpox, and other toxæmias, or may follow orbital or sinus disease, menstrual suppression, alcohol or lead intoxication; and occasionally is found in insular sclerosis and myelitis.

In *chronic retrobulbar neuritis* there exists with dimness of vision a small central color scotoma, particularly for red and green, the horizontal oval area in the visual field extending from the fixing point to the blind spot. The affection is found principally in persons using large quantities of tobacco, especially when combined with the use of alcohol. It is most frequently noted between 40 and 50 years of age, and has also been found in alcoholics who are not users of tobacco, and from toxæmia of lead, cannabis indica, stramonium, chloral, carbon bisulphide, iodoform, etc. The disturbance of vision is greater for near objects, and is more marked in bright light.

A form of retrobulbar neuritis similar to that of toxic origin appears as an hereditary affection, and is referred to as *hereditary optic neuritis*. It affects several members of a family, especially the males, and has been traced through several generations. The optic nerves show distinct pallor. Vision is reduced by a permanent central scotoma.

Hemianopsia (**hemianopia**) is a loss of one-half of the visual field of one or both eyes, due to a lesion in the optic chiasm, along the optic tracts, or in the visual centres in the occipital lobe. It does not include defects in the field caused by disease within the eyeball. The line dividing the seeing from the blind field is horizontal or vertical, or nearly so, and may cut exactly through the fixing point, or circumscribe this point by a small zone of preserved vision. (See Fig. 387, p. 733, Vol. II.)

The dividing line may have an oblique direction, but this is extremely rare, or only a sector, commonly a quadrant, of the field may be wanting.

Hemianopsia is classified according to the relative position of the blind portions of the two fields. It is *homonymous* if there is loss in the corresponding halves of each field; *bitemporal* if both temporal fields are blind, and

binasal when the nasal halves are lost. When the dividing line between the lost and preserved field is vertical, the defect is known as *vertical hemianopsia,* and when the dividing line is horizontal, the hemianopsia is *horizontal* or *altitudinal.*

Homonymous hemianopsia is the commonest form, and reveals itself as a defect in the right or left half of each visual field. For instance, in Fig. 152 the left half of each field is wanting, showing loss of function in the right half of each retina. If the right half of each field is lost the condition is *right lateral hemianopsia;* in loss of the left half of each field, *left lateral hemianopsia.* The seat of the lesion in homonymous lateral hemianopsia is in any part of the visual tract between the chiasm and the occipital lobe.

Bitemporal hemianopsia is a comparatively rare phenomenon, but one of great diagnostic moment when found. It manifests itself as a blindness

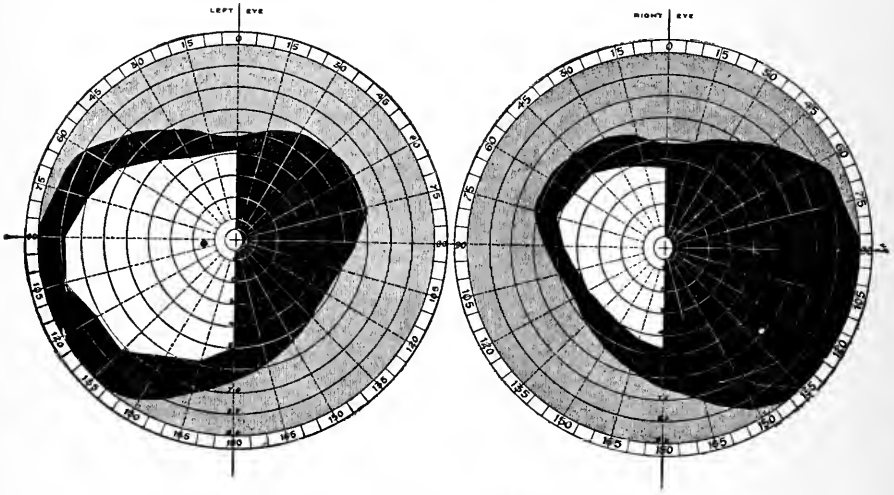

Fig. 152.—Diagram of perimetric charts of right lateral hemianopsia. The dark areas show loss of the nasal half of left and temporal half of right fields, with contraction of the preserved fields. The dividing line passes around fixing point.

of the outer or temporal halves of the visual fields, indicating suspended function of the nasal portions of each retina. It is caused by a lesion which destroys the function of the crossed fibres without affecting the uncrossed fasciculi. This may be a tumor, fracture, exostosis, aneurism, or disease of the blood-vessels. Loss of the two temporal fields is seen in acromegaly, although it is not a constant symptom, since the type of hemianopsia will depend upon the direction the pressure is exerted upon the chiasm and tracts.

Binasal hemianopsia, in which both the nasal fields are lost, is rare. If it is true that the crossed and uncrossed fibres of the optic nerve are mingled at the outer half of the chiasm, then a lesion of this structure cannot cause binasal hemianopsia. Shoemaker believes that this defect in the fields is due to an inflammation of the optic nerves.

Both upper or both lower fields may be wanting. In this condition, the lesion is, as a rule, at the chiasm, encroaching on it from above or below.

If the blind halves of the field have lost not only perception of form

and light, but also of color, the defect is *absolute;* if only recognition of color is lost, the hemianopsia is *relative.*

Hemianopsia as a Diagnostic Symptom.—In lateral hemianopsia the intracranial lesion is on the opposite side from the dark fields. If unassociated with motor or sensory symptoms, the lesion is confined to the cuneus, or the immediately surrounding gray matter; a lesion in one nerve tract, or in the primary optic centres, with symptoms of basal disease, would cause changes in the pupil, and possibly some affection of the nerve head could be recognized. Hemiplegia and hemianæsthesia are often present with lateral hemianopsia, indicating organic disease of the brain, the lesion being situated in the internal capsule. If right hemiplegia and aphasia are associated with lateral hemianopsia, an extensive lesion probably exists of the area supplied by the middle cerebral artery. A lesion of the posterior gray matter of the optic thalamus could produce lateral hemianopsia, with hemianæsthesia and ataxia of one side of body. A cortical lesion is usually associated with concentric contraction of the preserved fields, or is found in cases in which the light sense is preserved, but the color or form sense is abolished.

Hemianopic Pupillary-inaction Sign.—This is an important localizing sign in hemianopsia, and consists in carefully noting if the pupil reacts to a beam of light thrown upon the non-functionating half of the retina. It is an extremely delicate test to make, owing to the difficulty of restricting the beam of light so that it shall illuminate the non-acting half of the retina without allowing any light to fall upon the seeing half. If the pupil reacts when the light is thrown upon either the blind or the seeing half of the retina, the lesion is back of the primary optic centres; but if there is no reaction when the light falls upon the blind side, but the pupil reacts when the light falls upon the functionating side, the lesion is in front of the primary optic centres, and in that position has affected the motor arc of the pupil. The test should always be made in a well-darkened room, with barely sufficient light to conduct the examination, and should be confirmed by a second observer before basing a diagnosis on its apparent presence. Owing to the difficulty of absolutely demonstrating the presence of the reaction it is of limited value. The hemianopic prism phenomenon of Wilbrand is regarded by many observers of more usefulness. It consists of placing a strong prism before one eye, so that the image of a small piece of paper on a blackboard is thrown upon the blind half of the retina. The observer notes whether the eye makes a compensatory movement when the prism is quickly placed before the eye, and a movement in the opposite direction when the prism is removed. The other eye is bandaged in making the tests.

Hysterical Amaurosis.—The diagnosis of visual defects due to hysteria is sometimes difficult, although healthy eye-grounds and pupils normally reacting to light would point strongly to hysteria. Cases of hysteric blindness have been reported, however, in which light failed to have any action on the pupil.

If unilateral blindness arises suddenly, following fright, emotional excitement, slight injury, or menstrual pain, hysteria may be suspected. While the defect may be bilateral, it is more often unilateral. It is not uncommon to find, associated with the ocular symptoms, other disturbances of sensation, such as hemianæsthesia of the skin, cornea, or conjunctiva. If

the amaurosis is restricted to one eye, under some conditions it may be transferred to the other temporarily; and, again, the unilateral character of the affection may entirely disappear in binocular fixation, as proved by the diplopia if a prism of sufficient strength to prevent normal fusion is placed before one eye.

Not only may the vision be reduced in hysteria, but changes in the peripheral field are common. The contraction in the field is usually equal in the different meridians, and is often of the tubular type, in which the limits of contraction remain the same, no matter what distance the test object is removed from the eye. The field for colors likewise shows concentric contraction, or the limits of one color may overlap that of another, or there may be a complete reversal of the colors. In some instances the so-called fatigue fields are found, in which the limits constantly change during the examination or vary according as the test object is carried from the temporal to the nasal side, or *vice versa.*

Optic Neuritis.—Inflammation may affect the optic nerves at their intra-ocular portions (papillitis) or in their course in the orbit (retrobulbar neuritis). Under the term *hyperæmia of the nerve head* is included a type of optic-nerve irritation in which the disks become of dull red color, the surface and margins veiled, and the lymph sheaths of the vessels prominent. It is seen in refractive error, particularly hyperopia and hyperopic astigmatism, after long-continued exposure to intense light or heat, in some types of inflammation of the uveal tract, in orbital and sinus disease, in chronic insanity, and from toxic agents.

Papillitis. —Optic neuritis may be manifest as a true inflammation of the nerve tissue, a swelling of the intra-ocular ending of the optic nerve, or as a descending neuritis. The changes in the optic-nerve head may range from a decided redness, moderate swelling, and blurring of the margins, to an intense rounded protrusion of the disk from inflammatory exudation, reddish gray in color and sloping down into the surrounding retina, the retinal arteries shrunken, and the veins full and tortuous and covered in by infiltration or ending in numerous hemorrhages. Upon subsidence of the inflammation the nerve head becomes grayish white in color, the œdema subsides, and the extent to which the pressure has affected the nerve-fibres is shown by the degree of optic atrophy that follows.

A moderate degree of papillitis, associated with hemorrhages throughout the retina, few changes in the vessels, and spots of fatty degeneration of the retinal elements, is described as *neuroretinitis,* and is the type most frequently found in association with renal disease. The intense swelling of the papilla, with exudation and tortuosity of the veins, is termed *choked disk* or *papillœdema,* and is the usual type found in certain forms of brain tumor.

The neuritis may be due to affections of the orbit, such as fracture, orbital tumors, purulent cellulitis, and sinus disease. Intracranial causes are tumors, syphilis, abscess, and meningitis. Tumors of the brain cause about eighty per cent. of the cases of choked disk, with cerebral syphilis about ten per cent.

The situation of the intracranial portion of the optic nerve tracts at the base of the brain renders them particularly liable to implication in inflammations of the basal portion of the meninges and to the pressure of

tumors and abscesses. In children, tubercular meningitis is usually accompanied by swelling of the optic disk. The absence of affections of the optic nerve does not preclude the presence of a new growth in the brain, although when the base, and particularly the cerebellum, is the seat of a neoplasm, swelling of the optic disk is almost always present. Double optic neuritis of high degree, rapidly progressive, and accompanied by marked exudation in the nerve and surrounding retina, usually indicates a tumor of the cerebellum, while one of slower growth, less intense, and either unilateral or considerably greater on one side than on the other, is seen in neoplasms of the cerebrum. In about one-third of all the cases there is associated palsy of the ocular muscles, usually the sixth or third nerves, but these palsies occur more frequently in brain syphilis than in brain tumor.

The "stellate figure" in the macula, which is seen in a large proportion of the cases of renal retinitis, is not uncommon in the intense papillitis of brain tumor. Tumors or abscesses of the frontal region or those arising from the meningitis rarely cause optic neuritis, although swelling of the optic disk may occur. A growth in the pituitary region may cause inflammation of the optic nerves, but it is more frequent to find simple atrophy.

Apart from the intracranial causes, optic neuritis may occur from general infections. These are in the nature of a toxin, occurring in such diseases as influenza, syphilis, malaria, rheumatism, erysipelas, and many of the exanthematous and continued fevers. Lead and alcohol may also cause inflammation of the optic nerve, and the same process is seen in anæmia, loss of blood, sunstroke, and after violent exertion. Syphilis may cause a primary neuritis or act secondarily through gumma of the brain or meninges.

Unilateral optic neuritis may be due to orbital or sinus disease, and in rare instances to cerebral tumor, in which the neuritis occurs on the side of the neoplasm. The inflammation of the retina and optic nerve of nephritis and certain constitutional disorders is often unilateral, but with the progress of the systemic disease the inflammation attacks the other eye.

Central vision is usually unimpaired even in intense papillitis during the acute stage, and if defects in vision occur they partake of the nature of defects in the visual field—an enlargement of the blind spot, concentric contraction, and inversion of the color fields, the latter occasionally preceding changes in the optic nerve.

Retrobulbar neuritis has been considered under Amblyopia.

Optic-nerve Atrophy.—Degeneration and atrophy of the optic nerves may be *primary*, when there has been no previous inflammation or swelling of the papilla, or *secondary*, if preceded by previous optic neuritis or due to affections of the retina and choroid. In both forms there are changes in the color of the disk, varying from a gray to grayish white, with the edges usually clear and distinct in the primary forms, but veiled in the secondary.

Primary atrophy is more frequently associated with spinal disease, particularly locomotor ataxia, in which it usually appears before the ataxic symptoms. It is also found in insular sclerosis, paralysis of the insane, and occasionally in lateral sclerosis. It may occur as a result of excessive hemorrhage from the stomach, uterus, or intestines, in the toxæmia of fevers, alcohol or lead poisoning, in chronic malaria, syphilis, and diabetes, in fractures of the base, and in deformities of the skull. Hereditary optic-nerve atrophy

appears in early adult life, with a short interval between the involvement of the second eye. After several months the optic nerve shows distinct pallor, with most frequently a permanent central scotoma.

Secondary or Consecutive Atrophy.—The contracted retinal arteries, the dilated and tortuous veins, and the veiling of the surface and edges of the optic nerve point to a previous papillitis. Extensive retinal and choroidal disease also results in atrophy of the nerve, as will pressure upon the nerve-fibres by an aneurism, tumor, or exostosis.

Retinitis.—The retina is implicated in disease affecting the intra-ocular end of the optic nerve, and also from extension of disease from the ciliary body and choroid. The inflammation is associated with œdema and exudation, reduction of vision, especially under reduced illumination, hemorrhages either in the fibre layer or deeper, small-cell infiltration, and tortuosity of the retinal vessels, with changes in their calibre. Congenital tortuosity of the retinal vessels, especially of the veins, is frequently seen. In individuals with congenital heart lesions the retinal veins are often dilated and tortuous, and the arteries are nearly as dark in color as the veins. Small hemorrhages may be found near the optic nerve or in the macula.

Retinitis, like iritis and irido-cyclitis, may be considered in the majority of instances as a manifestation of some toxic process, such as is seen in many constitutional disorders, alterations in the condition of the blood and vessels, and infections. It may follow extension from other ocular structures. It is often divided for study as to etiology into syphilitic, diabetic, renal, traumatic, hemorrhagic, etc.

Syphilitic Retinitis.—The most common form of inflammation is associated with proliferation of the pigment layer, with marked changes in the choroid, and is associated with reduced vision, dust-like opacities in the vitreous, and night blindness. Hereditary syphilitic affections are seen in diffuse or circumscribed patches of retinal and choroidal atrophy with some pigmentation.

Diabetic Retinitis.—The small white dots with hemorrhages in the region of the macula and towards the optic nerve from the macula is the more frequent form. In some cases there are extensive areas of fatty degeneration with yellowish exudation in the region of the macula, with smaller areas and hemorrhages scattered through the retina. Both eyes are affected, sometimes with an interval between.

Albuminuric Retinitis.—The early changes appear as swollen and tortuous capillaries, congestion of the nerve head as indicated by a change in its color, with later the appearance of small round whitish dots and hemorrhages in the macula. The typical star-shaped figure is not always present. Usually both eyes are affected, but the signs of congestion and œdema may for a time only be in one eye, with implication of the other eye at a subsequent date. The above appearances are not found in every case of chronic renal disease, and probably not more than one-fourth of the cases of chronic renal disease exhibit nerve and retinal changes. The ophthalmoscopic pictures seen in pregnancy are similar to those mentioned above, and are due to the same toxins that produce the lesions in chronic renal disease. The differentiated diagnosis between the fundus changes in intracranial disease and kidney affections must be determined by other factors, including the

urine examination and general symptoms. The swelling of the optic nerve and the exudation are more intense in brain affections.

Retinal Hemorrhages.—Extravasation of blood into the retina may occur independently of any inflammation of the retina. It is usually the evidence of extensive vascular disease, or of organic heart affections. It occurs in the type of neuroretinitis associated with chronic nephritis, diabetes, and general arteriosclerosis; also in scurvy, purpura, polycythemia, marked anæmia, in the new-born suppressed menstruation, and in compression of the thorax. Embolism or thrombosis is associated with extensive hemorrhages from the retinal vessels. Retinitis with hemorrhages resembling those seen in renal disease are often present in simple anæmia and chlorosis. The position and extent of the hemorrhage determines the effect on vision.

Obstruction of the Retinal Vessels.—An embolism of the central artery of the retina is regarded as of rare occurrence, the general symptoms of occlusion of the artery or its branches being secondary to thrombosis or to obliterating disease of the walls of the vessel. Sudden blindness follows complete obstruction of the central vessel, whereas in plugging of one of the smaller vessels the blind area will correspond to the section of the retina supplied by the vessel affected. Prior to the attack there may have been periods of blurred vision for a considerable period, due to spasm of the retinal vessels. The fundus picture in embolism of the central artery shows a pallid disk, great contraction of the arteries, a clouding of the retina, and the appearance of a central red spot in the fovea. The affection is seen in valvular cardiac lesions, in endarteritis, and changes in the composition of the blood, although in about one-third of the recorded cases no cause could be found, the patients being in good health and later show no evidence of organic disease. A large proportion of those affected were females under thirty years of age.

Thrombosis of the central retinal vein is accompanied by rapid reduction in the vision until only the ability to count fingers at a few feet is preserved, with the ophthalmoscopic picture of large hemorrhages with later yellowish-white areas between them. In incomplete obstruction the disturbance of vision depends upon the extent of the involved retina. Secondary glaucoma is a frequent complication, requiring enucleation of the eyeball, as the disease is not favorably influenced by the usual forms of treatment by miotics or iridectomy.

Retinal Detachment.—Separation of the retina is seen in high degrees of myopia, following traumatism, effusion of blood or exudation beneath the retina, and from growths of the choroid. The detachment, which may follow direct injury, sudden strain as in vomiting, coughing, or lifting heavy objects, is sudden, and is accompanied with sudden loss of vision in that portion of the field of vision opposite to the detached area. In instances of slowly formed detachment, the vitreous becomes fluid and filled with floating opacities. The diagnosis of detachment is readily made with the ophthalmoscope, especially if the separation is of large size, the separated retina being thrown in folds of pearly gray color, over which the retinal vessels pass.

Arteriosclerosis.—A study of the changes in the retinal blood-vessels is of extreme importance as bearing on the early diagnosis of various phases of general arteriosclerosis. The early alterations in the retinal circulation

which should direct attention to general symptoms indicative of beginning sclerotic changes are tortuosity of one or more of the smaller arteries, the evidence of undue pressure of an artery at its point of crossing of a retinal vein, an increase of the light reflex of the arteries, and irregularity of the calibre of the veins. At first the vein is simply displaced in the direction of the arterial circulation, and its flow slightly obstructed; later the venous current is markedly impeded, and the vein greatly narrowed where the arterial pressure is exerted, and is distended on the peripheral side. These changes are rarely accompanied by sufficient fibrous thickening to cause white lines of perivascular inflammation along the vessel. As the vessel walls lose their elasticity, the impediment to the flow of blood results in tortuous vessels, the escape of fluid into the surrounding tissues, and retinal œdema. These conditions are not due to old age only, but to actual sclerosis of the vessels from disease. These early changes are difficult to diagnose except by a skilful observer.

The importance of early recognition of these ocular changes lies in their association with similar disease of the brain and kidney. There is no difficulty in determining by the ophthalmoscope the evidence in the eye-ground of well-advanced types of arteriosclerosis, but it is important that recognition of these signs should be made before the disease has reached a point where treatment is ineffectual. De Schweinitz called especial attention to the value of early recognition of the signs, even though they be only suggestive of angiosclerosis of the retinal vessels in persons who have reached the age at which vessel degeneration may begin to appear, and who consult the ophthalmologist for a change of reading glasses. These signs are "a corkscrew appearance of individual vessels, a slight thickening of the perivascular lymph sheaths, a beginning brick-dust appearance of the optic nerve-head, and a flattening of a vein against an artery or a bending in a curve of the vein overlying the artery." With these retinal conditions present the physician should carefully examine the cardiovascular system, and accurately test the arterial tension by approved means, and, should the tests confirm the retinal findings, institute appropriate treatment, which may save not only lesions of the eyes but of other structures, notably the brain, which, if they occur, may prove fatal.

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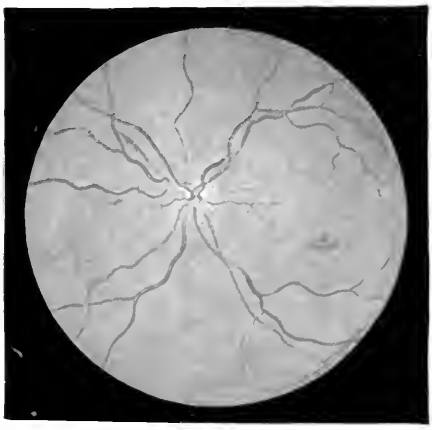

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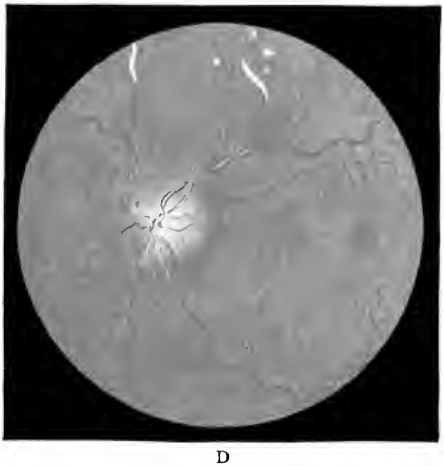

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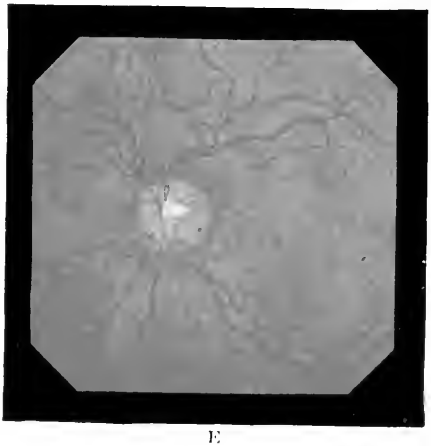

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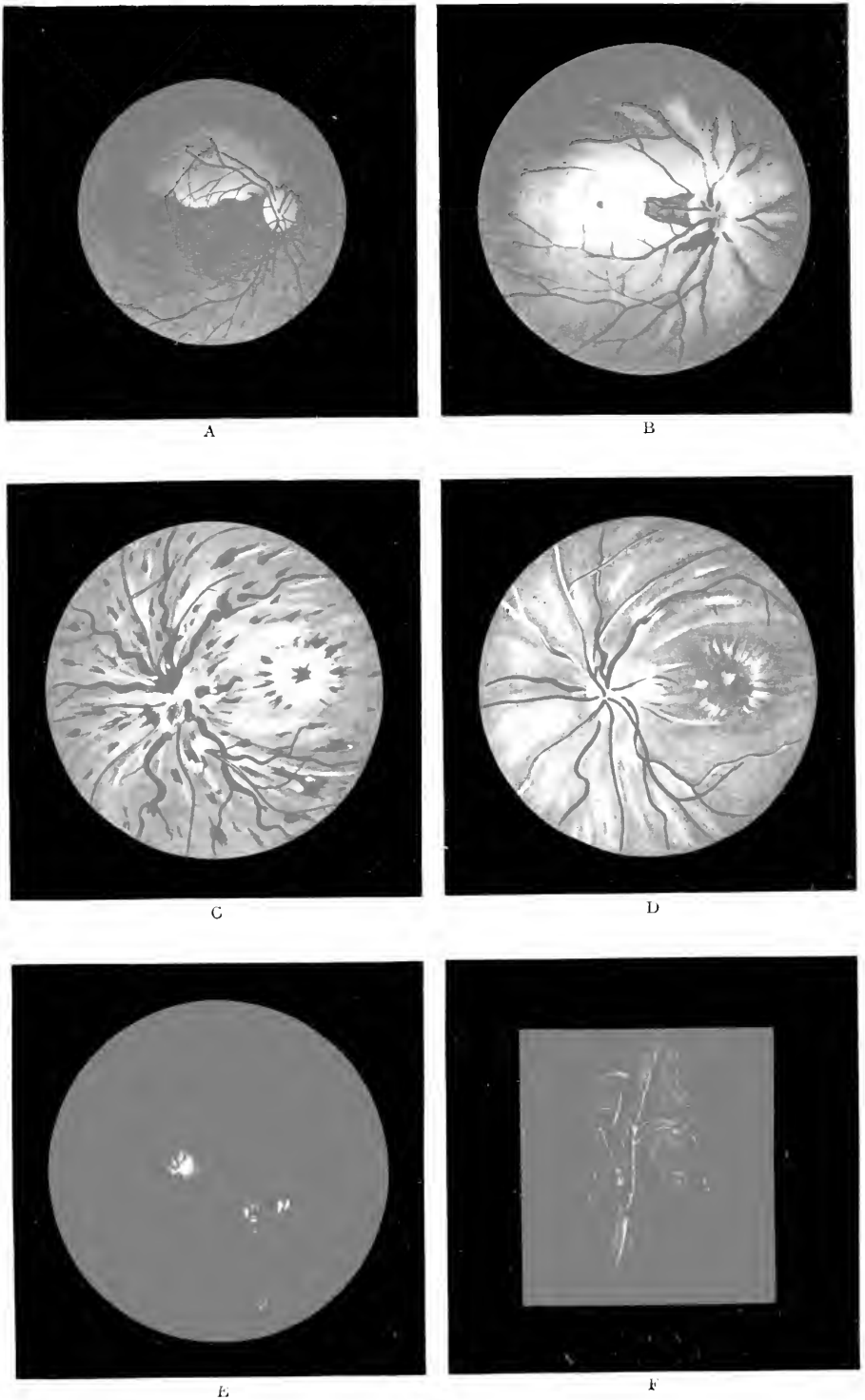
F

[CHANGES IN ARTERIOSCLEROSIS. After De Schweinitz.

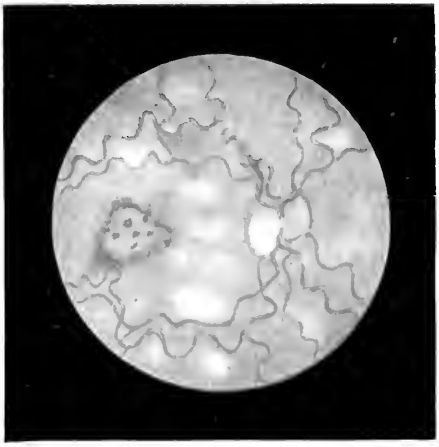
A, Normal fundus. B to F, successive changes occurring in arteriosclerosis, including pallid arteries (B), later assuming a silver-wire appearance (C); indented veins (B, C), afterward showing ampulliform enlargements (D, E); corkscrew capillaries (C, D); corkscrew arteries and veins (D, E); perivasculitis (C, D); sclerosis of vessels (E); œdema of disk (B, C, D, E), hemorrhages (C, D, E).]



[Changes in Retinal Vessels. After Wordemann in Posey and Spiller.

A, Embolism central artery, partial, affecting only inferior branch (Haab). B, Embolism central artery, total within nerve; a cilioretinal vessel supplies a small area of retina in which function is preserved (Wordemann). C, Thrombosis of central vessels from neuritis (Wordemann). D, Same case six months later, showing sclerosis and atrophy (Wordemann). E, Hemorrhages from retinal vessels (Magnus). F, Perivasculitis luetica (Magnus).]

A



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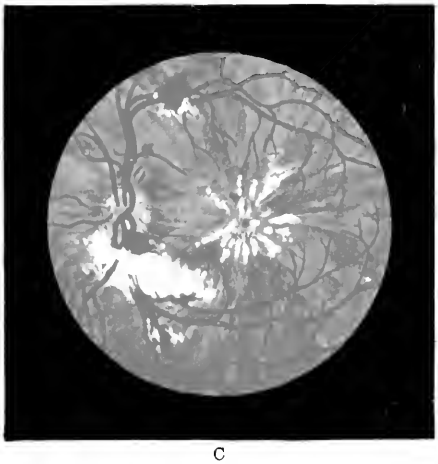

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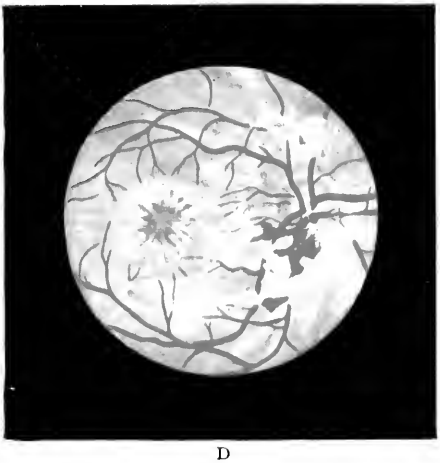

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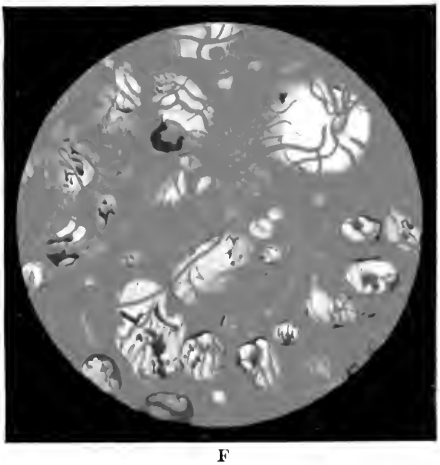


F

[Inflammations of the Retina.—After Wurdemann in Posey and Spiller.

A, Œdema in pernicious anæmia (Oliver). B, Leucæmic retinitis (Oliver). C, Albuminuric retinitis and neuritis of pregnancy (Wurdemann). D, Albuminuric retinitis in the negro (Wurdemann). E, Syphilitic retinitis (Haab). F, Atrophy of retina, choroid, and nerve following chorio-retinitis luetica (Oeller).]

XI

EXAMINATION BY MEANS OF RÖNTGEN RAYS.[1]

Röntgen made his discovery in 1895. It was a new kind of energy, its true nature unknown, hence he termed it X-ray. Within a month from the time of his discovery he disclosed three facts which have made these rays the most important diagnostic agent in the field of medicine, viz., that they penetrate all objects to a degree somewhat in direct proportion to the atomic density of the object; that they have actinic properties similar to ordinary light in their action on photographic plates; and that they excite fluorescence in certain substances, notably barium platinocyanide and tungstate of calcium. For example, the rays will penetrate all of the structures of the hand, but they will pass through the soft parts more freely than through the bony parts; therefore, if the rays after passing through the hand are permitted to act upon a photographic plate, which is protected from other forms of light, the various portions of the hand will record their respective densities, and after the process of development the plate will become a permanent record of shadows, appreciable to the naked eye. Such a record is termed a skiagram, a radiogram, or preferably a röntgenogram. If a screen covered with an emulsion containing crystals of barium platinocyanide or tungstate of calcium be substituted for the photographic plate, and other forms of light be excluded, the Röntgen rays, in passing through the hand, will be obstructed according to the density of the various parts of the hand and one will see immediately the shadows on the fluorescent screen. Such a screen is known as a fluoroscope, or, better, a röntgenoscope. All Röntgen-ray examinations, then, are made by means of röntgenograms, or by means of the röntgenoscope. The diagnostic value of such examinations depends directly upon the technic and interpretative skill of the röntgenologist. In the hands of the inexperienced and untaught the results are often valueless or misleading; and, because of the modern powerful exciting apparatus, the patient is in danger of being injured.

The röntgenographic method of examination has a far wider field of usefulness than the röntgenoscopic. The röntgenogram is a permanent record, can be studied indefinitely, and shows much finer detail in shadows than can be seen by means of the röntgenoscope. The former is applicable to all parts of the body, whereas the latter has advantages only in the study of movable organs—*e.g.*, the thoracic viscera and the gastro-intestinal tract. Röntgenoscopic examination should be made only with the most protective apparatus and continued for as little time as possible, to insure safety to operator and patient.

Stereoröntgenograms give accurate depth relations and are most useful. To make stereoscopic röntgenograms it is necessary to have a plate holder which will permit of plates being inserted and removed without disturbing the patient or the X-ray tube holder. Two exposures are made, the focus tube being displaced two and one-half inches either laterally or longitudinally. All modern tube holders are supplied with automatic or easily adjusted dis-

[1] Contributed by Professor Menges as collaborator.

placement device. The resultant negatives are then viewed by means of a stereoscope. Reductions may be made from the original negatives and viewed by means of the ordinary hand stereoscope.

Examinations of the head should always be made stereoröntgenographically. In this way the diagnosis of lesions of the accessory sinuses, including the mastoid cells, is made with much accuracy. A sinus filled with pus is more nearly opaque to the rays than one containing air; even granulation tissue or retained mucus casts shadows distinguishable from the normal. The importance of this procedure becomes apparent when one considers the frequency of sinus or mastoid infection as complications or sequelæ to the infectious diseases. Intracranial lesions are shown by the effect they produce on the inner table of the skull or the sutures, or, if they possess a density greater than that of the brain substance, by the presence of foreign shadows. Any localized organic lesion of the cortex of the brain will produce changes in the inner table of the skull either by way of bone atrophy due to increased

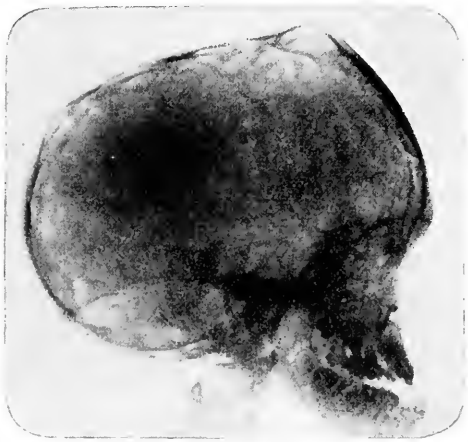


Fig. 153.—Convolution depressions very deep, due to early ossification of the sutures and moderate internal hydrocephalus. Child is a congenital syphilitic.

local pressure or by actual destruction of bone involved in the disease process. When intracranial pressure is increased by dilatation of the ventricles the convolutions of the brain soon produce impressions on the inner table which show clearly on the röntgenogram, and in the cases of children the skull sutures are shown to be more separated than normal. Acute external hydrocephalus causes the sutures to separate, but even the normal convolution impressions are absent. Disorders of the pituitary are manifested by some deviation from the normal in the röntgenographic appearance of the sella turcica. Diseases and abnormalities about the teeth may be shown clearly.

The thorax, because of the great contrast in density of its contained organs, is most accessible to Röntgen-ray study. One should become thoroughly familiar with röntgenograms of many normal chests before attempting to recognize the abnormal unless it has been carefully indicated by one who is skilled. Only in this way is it possible to appreciate the very early deposits of a tuberculous lesion or other infiltrating process, a thickening of the pleura,

inflammatory thickening or dilatation of the bronchial tubes, enlargement of the peribronchial glands, the extent of lung involvement, the presence and extent of pneumothorax or of pleural effusion, enlargement of the thymus gland, existence of substernal goitre, and variations from the normal in the

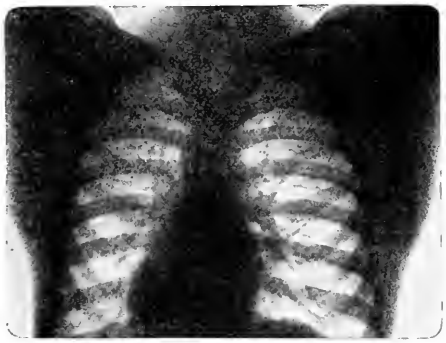


FIG. 154*a*.—Enlarged peribronchial glands. Lung shadows normal. (Viewed from the back.)

size and contour of the heart and large blood-vessels. Some of these conditions are diagnosed positively without other aid, but as a rule careful physical examination and clinical study are essential to correct differential diagnosis. For instance, the röntgenogram of enlarged peribronchial glands does not

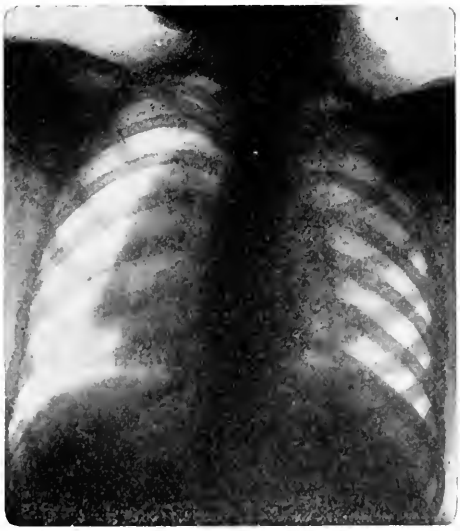


FIG. 154*b*.—Extensive pneumothorax on left side, slight displacement of heart. (Viewed from back.)

indicate whether the enlargement is due to tuberculous or other infection, and a history of measles, influenza, or other infectious disease is sufficient to limit the diagnostic value of the plate. Again, the infiltration of syphilis has much the same röntgenographic appearance as tuberculous infiltration, and so instances might be multiplied. All intrathoracic conditions except

mediastinal tumors, lesions of the circulatory organs, and those involving the diaphragm are best revealed by the röntgenographic method. The presence or absence of pulsation, and the excursus of the diaphragm can only

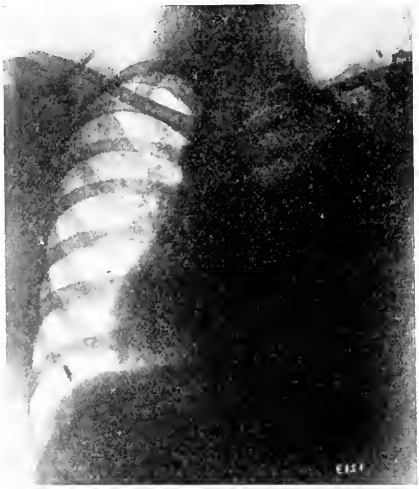


FIG. 155*a*.—Extensive pleura effusion on right side, very little displacement of heart Notice obliquity of upper leve of fluid (Viewed from back.)

be determined by means of the röntgenoscope. With the modern apparatus one can make a tracing which shows accurately the size of the heart and its relations to other structures, the size and extent of pulsation of an aneurism,

FIG. 155*b*.—Recurrent sarcoma of chest wall. Notice sharp outline of growth. (Viewed from back.)

the excursus of the diaphragm, etc. The size of the heart in its lateral and vertical dimensions can be shown on a röntgenogram if the exposure is made with the tube at a distance of seven feet or more. Such a picture is known as a teleröntgenogram.

Again, stereoröntgenograms are of the utmost importance in the study of lung lesions. Only in this way can one determine the depth and true size of isolated foci of disease or cavities. The earliest possible signs of pulmonary tuberculosis are minute areas of infiltration in the parenchyma and a matting or clumping of the very small branches of the bronchial tubes or bronchioles. Even in the normal chest these fine tubes can be traced to the very periphery of the lung if the röntgenograms are of excellent quality. The stereoscope enables one to see whether they proceed to the periphery discretely or whether they are matted together at points. The two plates must be exposed and interchanged in a period of time during which the patient can hold a full inspiration at complete rest. The tube should be displaced parallel to the longitudinal axis of the body. The patient may be in an erect

Fig. 156*a*.—Metastatic sarcomata of both lungs. (Front view.)

or horizontal position, but exposures should be made from both front and back. The erect posture has the advantage in that a lung cavity partly filled with pus will show an upper level.

Pneumothorax is readily distinguished from every other condition by the complete absence of lung shadow within the area of the plate covered by the air-containing space. Occasionally pneumothorax and pleural effusion coexist. In such cases the examination must be made with the patient sitting or standing, when a sharp transverse upper level of dense fluid shadow will be seen with a less dense pneumothorax area above it. On fluoroscopic examination waves can be set up and seen in the upper level of the fluid by percussing the chest wall. These waves cannot be produced in simple pleural effusion, and the upper level of fluid is obliquely downward and inward. Purulent and non-purulent effusions cannot be differentiated by means of X-ray. Encysted effusions are determined by their more or less rounded

form and by the fact that gravity does not determine the inferior boundary. They may be interlobar, or between the parietal and visceral pleura.

Localized or general thickening of the pleura is determined by comparative study of the unaffected portions, the thickened areas producing shadows considerably more dense than normal areas, but not so dense as an area of pleural effusion.

Röntgen-ray examination of the entire alimentary tract is made possible by the ingestion or injection of a substance opaque to the rays, or in certain instances the introduction of air which permits the rays to pass more freely. Bismuth oxychloride, bismuth subcarbonate, and barium sulphate (specially prepared) are the opaque substances in general use. Here the röntgenoscopic method is essential.

Diseases of the œsophagus are distinguished by their effect upon its lumen and course. A diverticulum will retain a portion of the bismuth and

Fig. 156*b*.—Larger aneurism involving ascending transverse and descending arch. (Viewed from back.)

cast a shadow of localized, pocket-shaped enlargement. Organic narrowing of the lumen is apt to be irregular, whereas spasmodic stricture is usually abrupt and smooth in outline. The former is constant, the latter intermittent, relieved by antispasmodics, and not accompanied with marked loss in weight. The œsophagus is best seen when the rays pass obliquely through the chest. The calibre of a stricture can be determined by having the opaque mixture of such viscidity that it will pass slowly under swallowing pressure from above through the narrowed portion.

The wide variations in size, shape, position and relations of the gastro-intestinal tract, within normal limits, make the Röntgen-ray study of these organs a task of large proportions. The variations in diseased conditions are even greater, and then, too, the stomach is reflexly influenced by disease in adjacent organs. Broadly speaking, the normal stomach is one which lies with its upper two-thirds in the upper left quadrant of the abdomen, with the lower third crossing the median line so that the pylorus is from one-half

to two inches to the right of the median line, and above the level of the umbilicus. The greater curvature is the lowermost portion, extending from the level of the umbilicus to two inches below this level. It fills gradually

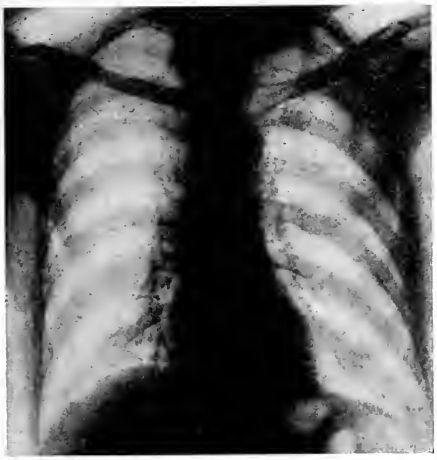


FIG. 157*a*.—Small particles of bismuth in the lower right bronchial tubes. There is communication with the œsophagus due to carcinoma.

from above downward so that the entire lesser and greater curvatures can be seen with a comparatively small quantity of opaque meal of thick fluid consistency, and enlarges throughout when completely filled, the posture being

FIG. 157*b*.—Organic stricture of œsophagus. Note bismuth in dilated portion above the stricture and irregularity of outline at the point of obstruction. (Oblique view.)

erect. The peristaltic waves begin at the junction of the upper and middle thirds, shallow at first, but become deeper as they approach the pylorus, where they end in the closed pylorus. The waves recur at intervals of about twenty seconds, and are deeper on the greater curvature than on the lesser

curvature until they approach the pylorus. The entire opaque meal leaves the stomach in from four to six hours.

In the athletic type of person with tense muscular abdominal walls the stomach is usually high, so that even the greater curvature is from one to three inches above the level of the umbilicus, and the long axis becomes more oblique with relation to the long axis of the vertebræ. The peristaltic waves are proportionately more vigorous and the emptying time less than the average normal stomach.

The opposite of this type of normal stomach is that in the individual whose abdominal walls are weak and relaxed. The greater curvature reaches three inches below the level of the umbilicus, while the pylorus is at or a

Fig 158*a*.—True ptosis of stomach.

little above the level of the umbilicus and more nearly in the median line. The peristaltic waves are lessened in vigor and the normal emptying time is about six hours.

When the greater curvature reaches a still lower level it is due either to dilatation or extreme atony, and the pylorus remains at the normal level, or, if it is to be considered true ptosis, the pylorus is at a lower level near the median line, and the greater curvature may extend into the pelvis. The ptotic stomach is usually more or less dilated. The emptying time of the dilated, atonic, or ptotic stomach is from seven hours to indefinite.

Röntgen-ray diagnosis of organic gastro-intestinal lesions should be based very largely on direct evidence; that is, the defect should be demonstrable either on the fluoroscopic screen or on sensitive plates, and often both are essential. Functional disorders and those due to faulty position or musculature require fluoroscopic study combined with palpation. Much of the evidence is direct, but indirect evidence is also of great importance in this class of conditions.

Ulcer is probably the most common of the organic stomach lesions. In the early or acute stages when only the mucosa is involved the X-ray findings are largely inferential. Deep spasmodic contraction is seen on the greater curvature, making a sharp incisure in the shadow of the opaque meal. The ulcer is usually on the lesser curvature at the level of the incisure. Incisure may occur as a reflex phenomenon, but will relax after the administration of antispasmodic drugs. In ulcer cases the incisure may remain for hours or recur intermittently. There is delay in emptying the stomach due to spasmodic condition of the pylorus. There is no point of tenderness, but deep pressure may excite a violent contraction, and the incisure will appear in spite of antispasmodics.

When an ulcer has penetrated the mucous and muscular coats a definite and constant deformity occurs in the shadow of the filled stomach. The deformity has the appearance of being a small diverticulum. Spasmodic

Fig. 158*b*.—Marked ptosis of colon. Same case as Fig. 158*a*

contractions may occur opposite the ulcer, but are not constantly associated. If the ulcer has healed in part there will be a more or less complete "hour-glass" contraction. The peritoneal coat may become perforated, and then small particles of the opaque meal can be seen isolated from the stomach shadow, and there is usually almost complete "hour-glass" formation due to cicatricial tissue contraction. There is usually a point of tenderness in deep ulcer. When ulcers occur near the pylorus it requires very careful observation to detect them in the early stages. The peristaltic waves on the lesser curvature are apt not to reach the pylorus, and there is retention. If the ulcer heals partly and contracts so as to produce mechanical obstruction, retention becomes more prominent and dilatation of the stomach follows. Vomiting is a rarely-associated symptom.

Carcinoma of the stomach can be diagnosed positively only when of sufficient extent to produce a permanent defect in the outline of the stomach

shadow. Then the distinguishing feature is the irregular punched-out appearance of the defect. Pyloric spasm is not present and the stomach empties more rapidly than normal if the pylorus is not obstructed. Vomiting and loss of appetite and weight are common symptoms when the lesion is at the pylorus, and hæmatemesis appears when the mucosa is destroyed. It is surprising, however, to what extent the stomach may be involved before symptoms appear when the pylorus is not involved.

In any lesion of the stomach involving the peritoneal layer adhesions form and the stomach cannot be displaced at such points by palpation.

Reversed peristalsis is occasionally seen, and so far as we know is always associated with some organic lesion of the stomach.

Fig. 159.—Carcinoma of pyloric end of stomach three hours after taking opaque meal. Almost complete obstruction of pylorus, very little dilatation. Exposure made obliquely to isolate stomach shadow from spine.

Pyloric spasm and consequent retention occurs reflexly from disease in other organs, such as the gall-bladder, kidneys, or appendix. In such cases further X-ray study is necessary, particularly during deep palpation.

In duodenal ulcer the most accepted signs are: irregularity in outline of first portion (where the vast majority occur); marked gastric hyperperistalsis; rapid emptying of the stomach during the first hour in non-obstructive cases; retention of bismuth in the stomach after six hours in obstructive cases; fixation of duodenum in late cases or in gall-bladder disease with adhesions to the duodenum; and occasionally sensitive pressure point over the duodenum.

In the intestines obstructive lesions are determined by the presence of dilatation of the proximal portion and delay in passage of the bismuth. The colon is to be studied by its action on the ingested meal as well as by injection. All of the usual chemical tests for acidity, blood, etc., should be made prior to the Röntgen-ray examination, as they sometimes influence the interpretation of certain findings. A complete clinical history is also an essential adjunct.

In diseases of the gastro-intestinal tract that are not locally destructive

or due to new growth the Röntgen-ray examination is of value in confirming the facts of relation to other viscera, and not infrequently showing lesions elsewhere which produce symptoms referable to the stomach. Kidney stone

Fig. 160.—Congenital dilatation of the colon. (Viewed from back.)

is often responsible for gastric symptoms which mask or exceed the renal symptoms.

Enlargement of the liver can be shown, gall-stones are demonstrable

Fig. 161.—Stone in right kidney.

in about fifty per cent. of the cases, and in slender patients the gall-bladder distended so that it projects below the liver can occasionally be shown. The size of the spleen can be determined, especially if the stomach and colon be distended with air or gas.

The urinary organs are studied by means of the röntgenographic method only. The röntgenogram should show enough of the outline of both kidneys to establish approximately the size and position in patients of one hundred and sixty-five pounds or less. This is due to the fact that the cortical portion of the kidney is more dense than surrounding structures, especially in the region of the lower poles. The pelves of the kidneys, the ureters, and the bladder do not cast distinguishable shadows. However, the outline of the entire urinary tract may be shown on the röntgenogram after the injection of a solution of colloidal silver, which is more opaque to the rays than the tissues. By this procedure one can differentiate between normal pelvis, hydronephrosis, tumors of the kidney, and abscess of the kidney if it drains into the pelvis. The shape of the kidney pelvis as shown on the plate is characteristic in each. Dilatations and kinks in the ureter can often be shown in the same manner. Such examination requires the assistance of an expert in ureteral catheterization. Renal, ureteral, and vesical calculi are readily demonstrable except those composed purely of uric acid. This variety is occasionally found in the bladder.

Diseases of the bones and joints which cause absorption, destruction, or proliferation of lime salts or cartilage lend themselves most aptly to röntgenographic study. In children the development of the skeletal structures is readily portrayed. The extent of the disease as well as its nature is usually shown, hence a prognosis as well as diagnosis may be more accurately made.

The value of the Röntgen-ray method of examination in excluding suspected conditions is scarcely less than in determining existing conditions.

The knowledge of the expert is as necessary to the explanation of skiagrams as to the making of them and it is always to be borne in mind that X-ray pictures are to be interpreted as shadows and not as lesions.

PART III.

OF SYMPTOMS AND SIGNS.

I.

GENERAL CONSIDERATIONS.

Symptoms and Signs.—The clinical phenomena of disease are divided into two general groups: (a) subjective, those obtained by inquiry, and (b) objective, those learned by observation. The former are known as SYMPTOMS, the latter as SIGNS. These may be general, as fever, debility, or emaciation, or local, as pain, dyspnœa, or dulness upon percussion. The dividing line between symptoms and signs is not well defined. Pain and nausea are symptoms of which nothing can be learned by observation alone; an endocardial murmur or pleural friction rub, signs concerning which the patient can give no personal account; while retching, cough, and palpitation partake at once of the nature of both signs and symptoms and may be referred to the one or other group of clinical phenomena according to the point of view from which they are regarded. SYMPTOMATOLOGY is that department of medical science which has for its object the consideration of the symptoms of disease; SEMEIOLOGY (*σημεῖον*, a sign), that which has for its object the consideration of the signs of disease. Just as symptoms and signs are not always to be closely distinguished, so the scope and subject matter of these sub-sciences of medicine largely overlap, and symptomatology and semeiology are frequently used interchangeably. Pure symptoms are limited in number as compared with signs, and, since they are wholly subjective and our knowledge of them is based upon the statements of the patient, who may, according to his temperament or for purposes of his own, either unintentionally or wilfully misrepresent them, they are of far less value in diagnosis than signs. Symptoms, which have their origin in the deranged sensations of the patient, stand in contrast to signs, which are dependent upon changes in organs or tissues. For this reason qualifying adjectives are sometimes employed, and we speak of rational symptoms and physical signs. It is customary, however, to apply the word symptom to many of the objective phenomena of disease.

SYNDROME is a term used to designate a set of concomitant symptoms, especially the concurrence of a group of symptoms not indicating a disease with well-determined anatomical lesions, as for example fever, seasickness, and astasia-abasia; a SYMPTOM-COMPLEX; a SYMPTOM-GROUP.

Diseases upon one basis of classification are divided into constitutional or general, namely, those in which the organism as a whole reacts to the pathogenic influence; and local or organic, in which the lesions involve primarily or chiefly a viscus or an anatomical tract. General symptoms

are often the expression of a local disease and local symptoms the expression of a general disease; thus emaciation, pallor, and a profound cachexia attend the progress of carcinoma of the stomach, while tenderness in the right iliac fossa, diarrhœa, and tympany are symptoms of enteric fever.

II.

APPEARANCE; TEMPERAMENT AND DIATHESIS; FACIES; FORM AND NUTRITION.

APPEARANCE.

The general appearance of the patient when first seen forms the groundwork for the study of his present condition—the *status præsens*. Whatever knowledge may be subsequently obtained of the facts in the case, the general appearance constitutes the point of departure for the direct diagnosis. In the successful clinician the habit of observing and noting its various details with great rapidity is cultivated in a high degree. The facial expression, state of nutrition, movements and attitude, mode of speech, and mental condition are at once observed. An opinion is formed as to what manner of man the patient is. Information as to his social position, occupation, and habits may be learned from his dress: Is he neat or slovenly? Are his clothes buttoned awry? Is his collar loose to make room for swollen glands or a goitre? Do his trousers show the white stains of diabetic urine? Has he the tabetic or steppage gait or the festination of paralysis agitans? Has he the flushed face with dilated venules and the trembling tongue of the alcoholic, or the enlarged girth and the waistband lengthened with a loop of string, of hepatic cirrhosis with ascites? The hands tell a story of their own. We note at a glance that they are white and soft and the finger-nails are clean, as occurs mostly, but not always, with men of leisure and professional men; that they are large and callous, as in those who follow the sea; coarse, sunburnt, and freckled, as in the farmer; or that they bear the oil and grime of the mechanic who has hurried from his work. The trembling hand of alcoholism, the pill-rolling movements of paralysis agitans, the nodules and tophi of gout, the deformities and relaxed ligaments of arthritis deformans, and the spade-like hands of myxœdema tell their own tale.

The diagnosis may sometimes be made at a glance. The flushed face, hurried breathing, unilateral movement of the chest, and rusty sputum of pneumonia scarcely demand the additional data of chill, crepitant râle, and percussion dulness; nor the intense headache, opisthotonos, vomiting, herpes, and petechiæ the history of sudden onset or the epidemic prevalence of cerebrospinal fever; while the paroxysm of whooping-cough is in itself diagnostic. The diagnosis thus made cannot, however, be called intuitive. In truth there is no such thing as an intuitive diagnosis. Before a conclusion is reached, however brief the time, the clinician, usually without being aware of his mental processes, has been weighing and sifting the symptoms

and assigning to each its proper value and importance. Such a diagnosis must in all cases be personally regarded as provisional and not announced, however tempting the circumstances, until sufficient facts for its full support have been obtained. There are pitfalls in the way of him who makes what in the language of the ward classes is called a "snap diagnosis." It is never complete; the pneumonia may be complicated with pericarditis. Such a diagnosis is sometimes altogether false; there are cases of enteric fever in neurotic individuals in early life that closely simulate the symptom-complex of meningitis—the so-called cerebrospinal form of enteric fever—and a child long convalescent from whooping-cough may under emotional excitement or when suffering from an attack of subacute laryngitis have paroxysms of cough with whooping.

TEMPERAMENT AND DIATHESIS.

Under the teaching of the French school great importance was at one time attached to temperament or diathesis as a condition of the hereditary constitution manifested in the general appearance of the patient. Less attention is paid to this subject now than formerly, but no very close observation is required to establish the fact that many individuals have in common peculiarities of physical and mental organization very different from those of others and that corresponding differences in general health and tendency to disease occur. Disregarding misleading refinements and combinations the following principal forms may be described:

The sanguine, sometimes called the Arthritic or Gouty Temperament.—The characteristics of this condition are well-developed bones and muscles, a fine skin, good hair, fair complexion, good nutrition, a general appearance of health and energy. The physiological processes are active, the digestion excellent, the bowels regular, the respiratory excursus large, the action of the heart regular, the pulse full and strong. In disposition persons of this temperament are cheerful and hopeful, hence the adjective "sanguine" often applied to them. Mentally they are active but of deliberate judgment and they do a large part of the world's work. They are especially liable to bronchitis and other catarrhal affections and to arteriosclerosis, and in advancing life prone to sclerotic changes in the valves of the heart, aneurism, angina pectoris, or apoplexy.

The nervous diathesis shows itself commonly in slender or undersized individuals who are often poor and irregular eaters, not well nourished. Such persons are alert and active but often incapable of sustained effort. The physiological processes are not always well performed. They often possess high intellectual ability and are subject to intense emotions. They are predisposed to derangements of the digestive apparatus and to headache from slight causes. They take things to heart, as the phrase goes, and are liable to break down under stress of work and worry. Neurasthenia, hysteria, migraine, and other functional diseases of the nervous system and insanity are common.

The Bilious Diathesis.—The complexion is dark, the hair dark brown or black, often coarse and oily, the skin shows a tendency to local pigmentation which varies in intensity, especially about the eyes and brow.

The appetite is irregular, often poor, fats and sugars are digested with difficulty, and the bowels are sluggish. The nutrition is not good, though women of this temperament often grow stout in middle life. They are subject to attacks of that form of gastrohepatic catarrh popularly described as biliousness and are often at such times faintly jaundiced. Nausea and headache are common. Such persons often lack energy.

The Lymphatic Diathesis.—The muscles are soft and flabby, there is very often a slight excess of subcutaneous fat. The skin is pallid, the physiological processes are sluggishly performed. Mentally such persons are dull and unresponsive. They are subject to enlargements of the subcutaneous lymph-nodes and are liable to chlorosis and other forms of anæmia.

The Strumous Diathesis.—The word struma, meaning scrofula or glandular tuberculosis, though still applied in another sense to goitrous swellings, has lost its significance in medicine and has almost disappeared from the modern literature. The term strumous diathesis has, however, a very definite significance and is applied to a bodily constitution unfortunately too common. The bony framework departs widely from the normal type. The chest is small and flat, the shafts of the long bones slender, their epiphyses enlarged and thickened. The musculature is undeveloped and soft. The appearance is characteristic, the head is large, the cranial bosses prominent, the forehead broad and protuberant, the lips full, the nose short and broad, its alæ thickened, the lower jaw small, the teeth carious, the complexion commonly fair, the hair fine and the eyelashes long. The nutrition is poor and the general appearance of such individuals delicate and frail. The cervical lymphatics are often enlarged. Such persons are subject to tuberculosis of the glands, bones, and lungs and miliary tuberculosis, and usually die at an early age. They are frequently the offspring of tuberculous parents. Whether the constitutional peculiarities which go to make up the so-called strumous diathesis are to be ascribed to profound derangements of nutrition, transmitted from tuberculous parents, or to a latent tuberculosis acquired in the earliest period of life cannot always be determined.

Cachexia is a term used to describe the ravages of certain chronic wasting diseases, especially untreated malaria, the graver forms of syphilis, and carcinoma, particularly when it involves the digestive organs. The cachexiæ present a combination of profound anæmia, extreme emaciation and debility, and a diffuse, faint, muddy pigmentation of the skin.

Dyscrasia is a depraved state of the system, especially of the blood, due to constitutional disease. In the words of Bristowe it is a general deterioration of health and functional disturbance caused by the deflection of nutrition. We speak of a tuberculous, malarial, syphilitic, or cancerous dyscrasia.

FACIES.

The appearance of the face is often of diagnostic importance. It frequently indicates the subjective sensations and not rarely the psychical condition. To say that a patient's expression is that of suffering, acute pain, anxiety, overwhelming illness, or that it is excited, dull or, stupid, is intelligible without further comment. The face is an index of the physio-

logical age of the patient. The gray hair, wrinkled brow, arcus senilis, and hanging folds of skin about the neck are very suggestive. They enable the clinician to compare the apparent age of the patient with his actual age as measured by years. The facies of fever patients is often characteristic. In the stage of excitement there is an intensification, in that of depression a blurring of expression, accompanied by a peculiar moist appearance of the eye, a feverish flush and often a very slight turgescence of the skin of which I shall speak more fully in a later paragraph. Equally characteristic is the facies of dyspnœa. Here also puffing or turgescence is present, sometimes marked, and there is cyanosis, and with these symptoms dilated nostrils, an open mouth, and hurried breathing. The flushed face and bright eyes that follow too much wine, and the bloated countenance with its blurred lines, dilated venules, thickened nose, acne, and trembling tongue of some forms of chronic alcoholism are sadly familiar. The characteristic change of the countenance seen in those about to die, especially in patients suffering from ileus, peritonitis, cholera, and similar diseases, is described as the FACIES HIPPOCRATICA. The changes are largely due to a diminution in the contents of the blood- and lymph-vessels and muscular relaxation. The skin falls back upon the bones, the lines of expression are more sharply defined than normally, the nose is sharp and pinched, the eyes sunken, the temples hollow. With the pallor there is some degree of cyanosis which gives the skin a leaden or faintly livid hue. The surface is cool and bathed with sweat.

FIG. 162.—Cerebrospinal fever, fourth day of attack. Fever facies; patch of herpes; retraction of head.—Municipal Hospital, Royer.

The appearance of the face in the following conditions is suggestive if not always characteristic:

Enteric Fever.—In well-developed cases the face is dull, expressionless, pallid, with a faint, dusky flush over the cheek bones, often slightly drawn. The eyelids are half closed, the lips pallid and separated, in neglected cases sordes may be seen upon the teeth. Such also is the facies of patients profoundly septic. It occurs in the so-called "typhoid state" and is seen in puerperal septicæmia, malignant endocarditis, infection after surgical operations, and the like.

Acute Peritonitis.—The expression is one of intense suffering, the face is pale and drawn, sometimes the upper lip is contracted so as to show the teeth.

Pneumonia.—A circumscribed flush of one or both cheeks may be seen; it may be bright or dusky. When one cheek only is flushed it is usually that upon the side of the pulmonary lesion. With this appearance in grave cases are associated the facial changes peculiar to dyspnœa. A similar appearance accompanies the symptomatic fever of phthisis—hectic fever. The flushing is, however, brighter and not so distinctly circumscribed.

It is in strong contrast with the pallor of the brow and neck. The striking appearance of such patients is intensified by expanding nostrils, hurried breathing, bright moist eyes, and an intense, often eager, expression.

FIG. 163.—Parotid bubo complicating enteric fever. —Jefferson Hospital.

Tetanus.—The facial peculiarity is startling. Under the action of the toxin of the disease all the muscles of expression are thrown into tonic spasm more or less intense at the same time, whereas under ordinary circumstances the varying moods are expressed by the contraction now of one set of muscles, now of another. The lips are parted and the corners of the mouth drawn up as in laughter or grinning, while other parts of the face and especially the brow are contracted and thrown into folds as though in grief or anger — RISUS SARDONICUS of the older writers.

The Exanthemata.—The appearance of the face in the eruptive infectious diseases is usually diagnostic. The diffuse uniform rash, often in strong contrast with the white border around the mouth, and the slightly tumid skin of scarlet fever, the turgid skin, coarse measly rash with its crescentiform arrangement and coryza in measles, the pock in different stages of development with its umbilicated vesicles or hideous pustules and crusts and swollen and disfigured features in the variolous diseases, and the pellucid hemispherical vesicles or crusts without areolæ, scattered singly or in groups about the brow or mouth in varicella, are characteristic.

FIG. 164.—Œdema in acute nephritis.—Jefferson Hospital.

Mumps.—The deformity, alike when it involves one or both sides, is characteristic. The swelling is at first limited to the region of the parotid gland, behind the jaw and below the ear, but the surrounding œdema sometimes assumes remarkable proportions. The lobule of the ear stands out at right angles to the side of the head. When the swelling advances upon the cheeks, the corners of the mouth are slightly drawn up. The parotid bubo which occurs in some cases of enteric

fever, pneumonia, septicæmia, and other grave infections superadds to the facies of those conditions a deformity somewhat like that of mumps. The overlying skin is usually of a dusky red color.

Renal Disease.—The striking appearance of the patient in some forms of acute nephritis and very commonly in chronic parenchymatous nephritis is of diagnostic importance. It is characterized by intense waxy pallor, marked œdema of the eyelids, and general puffiness of the face by which the lines of expression are to some extent impaired.

FIG. 165.—Facies in a case of adenoid vegetations of the nasopharynx.—Merrick.

Hepatic Disease.—In chronic diseases of the liver and especially in cirrhosis and gall-stone disease the facies is peculiar. The features are as a rule sharp, the face thin, conjunctivæ muddy, venules dilated, lips red, and skin slightly jaundiced or subicteroid—FACIES HEPATICA of the older writers.

Malaria.—The pallor, sallowness, and muddy conjunctivæ which are so commonly seen in intensely malarious districts are not without considerable value in the diagnosis of the cachexia of this disease.

Syphilis.—The face may show characteristic eruptions at different stages of the disease. The coppery flat papule or papulosquamous syphilide upon the temples and forehead—CORONA VENERIS—is common. The face of the patient under treatment may show not the eruption of the disease but the drug exanthem produced by the iodides. Babies suffering from inherited syphilis are usually pale, weazened, and wrinkled. They look curiously like miniature old men. They have snuffles and superficial excoriations about the angles of the nose and corners of the mouth.

FIG. 166.—Exophthalmic goitre.—Jefferson Hospital.

Rickets.—The frontal and parietal eminences are exaggerated and the top of the skull flattened, so that the head assumes an appearance of squareness and is sometimes spoken of as box-shaped.

Hydrocephalus.—The head is characterized by its spherical shape, great size, and protruding eyeballs, the result of depression of the orbital

plate of the frontal bone. The exophthalmus is sometimes so marked that the eyelids cannot be closed. The size of the head is often enormous, its diameter may reach 20 to 25 cm. in a child a few years old. The face on the contrary appears very small, its expression vacant and fatuous. The cranial bones are separated and exceedingly thin. The hair is scanty and the veins may be seen beneath the skin.

FIG. 167.—Leprosy.

Hypertrophy of the Tonsils and of the Adenoid Tissue of the Pharnyx.—As a result of habitual mouth-breathing the expression of the countenance gradually undergoes characteristic changes, the face becomes apathetic and vacant, the nostrils are narrow, the lips thick, and there is projection of the upper jaw and lip.

Myxœdema.—The face is "moon-shaped," swollen and flattened, the nose broad, the mouth coarse and large, the lines of expression obliterated. The skin is yellow, waxy, dry, and scaly, the hair thin and scanty, the cheeks and nose flushed.

Cretinism.—The face is large, the lips thick, the tongue large and protruded, the mouth open and drooling, the nose flattened, the skin pallid and waxy, the expression idiotic.

Acromegaly.—The bony hypertrophy is especially manifested in the supra-orbital arches, the malar bones, and in the projecting lower jaw. The forehead is receding, the nose is increased in size, its alæ distended, the eyelids enlarged and thickened. The cartilages of the ear are also enlarged so that very often the ears stand out conspicuously from the head.

FIG. 168.—Facial paralysis following cerebrospinal fever.—Jefferson Hospital.

Exophthalmic Goitre.—The protrusion of the eyeballs, sometimes so marked that the patient can no longer close his eyes, produces a remarkable change in the expression. In its lighter grades the air is that of surprise, but when the exophthalmus is marked the patient has a frightened or astonished look which is intensified by the characteristic tremor. The enlargement of the thyroid body as well

as the visible pulsation and venous distention add to the peculiar expression of the countenance in this disease.

Leprosy.—The development of leprous nodules upon the face and the thickening of the skin give rise to remarkable deformities. The chin, lips, nose, eyelids, and ears undergo peculiar and characteristic changes, in consequence of which the face assumes the appearance of a hideous expressionless mask. Baldness, loss of the eyebrows, lashes, and beard, and ulceration also occur. The disfigurement suggests the conventional face of the satyr or the lion and is often spoken of as leonine—FACIES LEONINA.

FIG. 169.—Hemiatrophia facialis.—After Hirt.

Nervous and Mental Disease.—In functional nervous diseases there are frequently changes in the countenance more easily recognized than described. The pallid, slightly drawn face of the neurasthenic with its habitual air of depression is familiar to the practitioner. These traits, somewhat intensified, are common in women broken down by frequent child-bearing and in those who suffer from disease of the pelvic organs—FACIES UTERINA. In hysteria the face usually remains free from the motor disturbances so common elsewhere, particularly in the lower extremities. There is neither spasm, paralysis, nor other constant characteristic save that it reflects, often intensely, the varying uncontrolled emotions of the patient. Equally without cause laughter succeeds tears or vivacity is followed by an air of sullen and dogged indifference. Central or peripheral disease of the nervous system may manifest itself in spasmodic twitching of the facial muscles. Mimetic spasm or convulsive tic consists of clonic contractions of the muscles supplied by the facial nerve. They are usually limited to the region about the eye or above the corner of the mouth. Sometimes they involve the greater part of one or both sides of the face. Similar contractions of the muscles of expression occur in children and are known as habit spasm. In peripheral facial palsy the affected side is smooth and motionless, the wrinkles of the forehead and the labionasal fold disappear, the corner of the mouth is lowered and frequently drools, and the mouth itself is slightly drawn toward the sound side. The eyelids

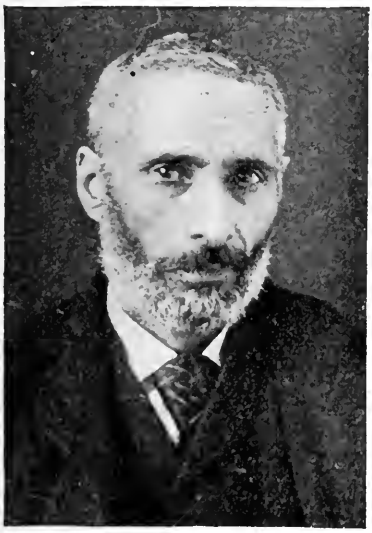


FIG. 170.—Paranoia, homicidal type.—Chase.

are motionless and can only be partly closed. The tears fall over the cheeks. The contrast with the opposite side is intensified upon efforts to smile or close the eye. When the paralysis is due to central causes the lower segment of the face is chiefly involved. In old cases, after contracture has taken place the mouth is drawn toward the affected side.

In organic disease tremor and paralysis are significant. Tremor of the lips and tongue occurs in chronic alcoholism. Fibrillary tremor is frequently associated with progressive palsy. In bulbar paralysis the lips are thin, compressed, and tremulous, the tongue is wasted and protruded with difficulty, and there is dribbling of saliva. In paralysis agitans the appearance of the countenance is very strikingly changed. The face has a curious stiff, expressionless immobility which has given rise to the name Parkinson's mask. There is often druling from the partially closed mouth and the lips and tongue frequently share in the general tremor. In general paresis local twitchings of the face, irregularity of the pupils, and slight tremulousness of the lips are suggestive. The rare disease facial hemiatrophy is a trophic neurosis affecting one side of the face, commonly the left. The soft tissues and bones are alike involved in the atrophic process, which is sharply limited at the mesial line. The eye is sunken and the corresponding half of the tongue and soft palate may be implicated.

The facies in disease of the mind is often characteristic. The depression of melancholia, the agitation and eagerness of acute mania, the alertness of chronic mania, the irregular contractions of the facial muscles in paresis, the fixed expression in paranoia with homicidal tendencies, the fatuous face of the imbecile, are well known to every student of psychiatry.

FORM AND NUTRITION OF THE BODY.

These are important factors in the problem of diagnosis. The normal of different individuals varies within wide limits. It is scarcely necessary to say that persons may be tall or short, stout or slight, fat or lean, without manifesting, even in wide ranges of difference in these respects, either the predisposition to or the symptoms of disease. Health consists in the ability of the individual organism to maintain its normal activities in the environment in which it happens to be placed. The somewhat pallid, hollow-chested and slender book-keeper often has good health and length of days while the clear-eyed, bright-skinned, deep-chested, well-developed athlete not infrequently breaks down in early middle life. Variations in these respects give rise to predisposition or constitute the indications of disease when they become excessive. We say that a man has a splendid or powerful physical development when the measurements of his body transcend the average, but the health of another who does not reach the average may be equally good. Not only anatomical structure but also physiological function are to be considered. Subtle hereditary tendencies, the value of which we cannot always estimate, and the shifting balance between the powers of the organism and the work which it is called upon to do engage our attention. To say that the physical organization is feeble, delicate, slender, robust, or muscular needs no further explanation. The condition of nutrition shows itself furthermore in the development and tonicity of

the muscles and especially in their relation to the amount of subcutaneous fat—panniculus adiposus. On the one hand a moderate amount of subcutaneous fat is not incompatible with excellent health, an excess is alike inconvenient and dangerous, and obesity constitutes a positive disease. On the other hand a spare man may be equally healthy, while rapid loss of fat is a suspicious symptom and emaciation an alarming sign of disease. In estimating the value of these conditions the hereditary tendencies, the occupation, the age, and the sex of the patient must receive due consideration. In young infants the panniculus is well developed. In infancy it often dwindles, only to increase again as puberty approaches. At this period it not infrequently again becomes excessive. In advanced life the fat commonly diminishes and the aged as a rule are spare. The panniculus is usually greater in women than in men and very often increases after the menopause. In most chronic diseases the nutrition is impaired because either sufficient nourishment is not taken on account of loss of appetite, or that which is taken is not assimilated. A high degree of emaciation attends diseases of the digestive organs and chronic febrile diseases, for example, carcinoma, especially carcinoma of the œsophagus or pylorus, enterocolitis with excessive diarrhœa, some forms of diabetes mellitus, phthisis, and enteric fever with repeated relapses. Wasting of fat is accompanied by wasting of muscle.

Weight.—The weight of the body and the amount of subcutaneous fat may be approximately estimated by inspection, but this method is uncertain and practically valueless in determining the progress of gain or loss. Accurate data can only be obtained by the use of scales at regular intervals and the preservation of the records for comparison. Small platform scales provided with a device for ascertaining the height of the patient are indispensable in the consulting room of the medical clinician engaged in the treatment of chronic cases. The automatic weighing machines found in public places in the cities are unreliable. Allowance must be made for the clothing and its variations in the different seasons, and when practicable the weight should be obtained shortly after the voidance of urine and an action of the bowels, and before a meal. Errors of two or three pounds may thus be eliminated, though for practical purposes in the long run slight fluctuations in the weight may be disregarded in the course of chronic disease. Many healthy individuals show an annual oscillation of several pounds in net weight, allowance being made for clothing, the minimum being reached in the spring or early summer, the maximum in the autumn or beginning of the winter. The body weight should be taken according to the requirements of individual cases at regular intervals of a week or longer. Daily observations are unnecessary.

The relation of the average body weight to the age and height of healthy individuals is set forth in the following tables:

Average Weight of Healthy Adult Males.—Hutchinson.

4 ft. 6 in. to 5 ft. 0 in.	92.26 lbs.
5 ft. 0 in. to 5 ft. 1 in.	115.52 lbs.
5 ft. 2 in. to 5 ft. 3 in.	127.86 lbs.
5 ft. 4 in. to 5 ft. 5 in.	139.17 lbs.
5 ft. 6 in. to 5 ft. 7 in.	144.29 lbs.
5 ft. 8 in. to 5 ft. 9 in.	157.76 lbs.
5 ft. 10 in. to 5 ft. 11 in.	170.86 lbs.
5 ft. 11 in. to 6 ft. 0 in.	177.25 lbs.

Infants, whether nursed or artificially fed, should be weighed at regular intervals of some days or a week. Important information is thus obtained not only as to the appropriateness of the food in kind and quantity but also as to the presence of assimilative disorders. The average normal weight of the newborn is, according to Uffelmann, in girls 3000 grammes, in boys 3500. During the first three or four days of life there is a decrease of from 220 to 300 grammes. After this there is in healthy children a progressive increase.

Average Daily Increase in Weight During the First Year of Life.—Gerhardt.

1st month	25 grammes.
2nd month	23 grammes.
3rd month	22 grammes.
4th month	20 grammes.
5th month	18 grammes.
6th month	17 grammes.
7th month	15 grammes.
8th month	13 grammes.
9th month	12 grammes.
10th month	10 grammes.
11th month	8 grammes.
12th month	6 grammes.

The weight index is the ratio of the weight of a given infant to the weight of the average normal infant of the same age.

Useful figures to remember are that the initial weight is doubled at 5 months and trebled at 15 months; also that the weight at one year is doubled at 7 years and that this weight is again doubled at 14 years (Rotch).

In infants and young children misleading inferences may be drawn from a consideration of the weight alone. There are some who are fat and flabby and not healthy. Such children are pallid, they lose and gain weight rapidly and have but little resisting power to disease. Then there are many who are bright and rosy, whose flesh is firm and solid, whose nutrition is good, who gain in weight normally and are not liable to the wasting diseases.

In the following table the comparative average weight of the sexes is shown. It will be observed that from birth until the sixth year the average weight in the two sexes is nearly the same. From this period for some years the weight of the female is considerably less than that of the male. About the age of puberty the difference becomes less marked, though the weight of the female is decidedly below that of the male.

Average Normal Weight in the Two Sexes at Different Periods of Life—Quetelet.

	Males.		Females.	
New-born	3.1 kgs.	6.82 lbs.	3.0 kgs.	6.60 lbs.
1st year	9.6 kgs.	19.80 lbs.	8.6 kgs.	18.92 lbs.
2nd year	11.0 kgs.	24.20 lbs.	11.0 kgs.	24.20 lbs.
3rd year	12.5 kgs.	27.50 lbs.	12.4 kgs.	27.28 lbs.
4th year	14.0 kgs.	30.80 lbs.	13.9 kgs.	30.58 lbs.
5th year	15.4 kgs.	33.88 lbs.	15.3 kgs.	33.66 lbs.
6th year	17.8 kgs.	39.16 lbs.	16.7 kgs.	36.74 lbs.
7th year	19.7 kgs.	43.34 lbs.	17.8 kgs.	39.16 lbs.
8th year	21.6 kgs.	47.52 lbs.	19.0 kgs.	41.80 lbs.
9th year	23.5 kgs.	51.70 lbs.	21.0 kgs.	46.20 lbs.
10th year	25.2 kgs.	55.44 lbs.	23.1 kgs.	50.82 lbs.
11th year	27.0 kgs.	59.40 lbs.	25.5 kgs.	56.10 lbs.
13th year	33.1 kgs.	72.82 lbs.	32.5 kgs.	71.50 lbs.

	Males.		Females.	
15th year	41.2 kgs.	90.64 lbs.	40.0 kgs.	88.00 lbs.
17th year	49.7 kgs.	109.34 lbs.	46.8 kgs.	102.96 lbs.
19th year	57.6 kgs.	126.72 lbs.	52.1 kgs.	114.62 lbs.
20th year	59.5 kgs.	130.90 lbs.	53.2 kgs.	117.04 lbs.
25th year	66.2 kgs.	145.64 lbs.	54.8 kgs.	120.56 lbs.
30th year	66.1 kgs.	145.42 lbs.	55.3 kgs.	121.66 lbs.
60th year	61.9 kgs.	136.18 lbs.	54.3 kgs.	119.46 lbs.
70th year	59.5 kgs.	130.90 lbs.	51.5 kgs.	113.30 lbs.

In cases of tardy or interrupted convalescence from an acute disease systematic observations of the weight of the patient at intervals of a week are of great use. A sudden arrest or decrease in weight may mark the development of a tuberculous process. Loss of weight is of great importance in the diagnosis of early phthisis. An arrest of the loss, still more a gain in body weight, must in most cases of this disease be regarded as favorable. There are, however, exceptional cases in which after a considerable steady gain in weight the tuberculous process suddenly makes grave or even fatal progress.

The weight is not in all cases merely an indication of the general nutrition and amount of fat. It is sometimes made up in considerable part of dropsical effusions, as in advanced disease of the mitral valve with rupture of compensation, of accumulations in the serous sacs, as in massive serofibrinous pleurisy or the ascites of cirrhosis of the liver, of the contents of cysts, as in enormous monolocular disease of the ovary, or of new growths of large size, as in the splenic tumor in leukæmia or sarcoma of the kidney in young children. In a dropsical patient the successful use of salines or diuretics may be followed coincidently with the subsidence of the anasarca by a reduction in weight amounting to many pounds in a few days. The tendency to accumulate excessive fat at middle life, especially in women after the menopause, must be regarded as pathological, and obesity, as has been said, constitutes a positive disease. The gain in weight occurs at the time of beginning decrease of muscular power, at the period of physiological involution. The individual must carry about a growing mass of inert fat with lessening ability on the part of the skeletal muscles to bear it and of the heart to carry on the circulation, and the disproportion between the burden and the ability to bear it increases with advancing years. Visceral fat accumulations occur, also fatty changes in the myocardium and vessels. Obese persons in early middle life bear the acute infections and especially enteric fever badly. Very often the fat accumulations are largely local, as in the mammæ, abdomen, and hips in women, or in the abdomen, abdominal walls, and omentum in men of sedentary life and given to the pleasures of the table.

III.

BONES; JOINTS; MUSCULATURE; POSTURE, ATTITUDE, AND GAIT; POSTURE AND MOVEMENTS OF INFANTS.

BONES.

The skeleton determines the stature and frame of the individual. As has been already pointed out the normal limits of variation in the measurements of the bony framework are very wide. Excess in either direction, as in gigantism or dwarfism, is pathological and has been ascribed to derangements of the functions of the pituitary body.

AVERAGE HEIGHT IN MALES AND FEMALES AT DIFFERENT PERIODS OF LIFE.—QUETELET.

	Males.		Females.	
New-born	50.0 cm.	20.00 in.	49.4 cm.	19.76 in.
1st year	69.8 cm.	27.92 in.	69.0 cm.	27.60 in.
2nd year	79.1 cm.	31.64 in.	78.1 cm.	31.24 in.
3rd year	86.4 cm.	34.56 in.	85.4 cm.	34.16 in.
4th year	92.7 cm.	37.08 in.	91.5 cm.	36.60 in.
5th year	98.7 cm.	39.48 in.	97.4 cm.	38.96 in.
6th year	104.6 cm.	41.84 in.	103.1 cm.	41.24 in.
7th year	110.4 cm.	44.16 in.	108.7 cm.	43.48 in.
8th year	116.2 cm.	46.48 in.	114.2 cm.	45.68 in.
9th year	121.8 cm.	48.72 in.	119.6 cm.	47.84 in.
10th year	127.3 cm.	50.92 in.	124.9 cm.	49.96 in.
15th year	151.3 cm.	60.52 in.	148.8 cm.	59.52 in.
20th year	167.0 cm.	66.80 in.	157.8 cm.	63.12 in.
25th year	168.2 cm.	67.28 in.	157.4 cm.	62.96 in.
30th year	168.6 cm.	67.44 in.	158.0 cm.	63.20 in.
40th year	168.6 cm.	67.44 in.	158.0 cm.	63.20 in.
60th year	167.6 cm.	67.04 in.	157.1 cm.	62.84 in.
70th year	166.0 cm.	66.40 in.	155.6 cm.	62.24 in.

There is a constant relationship in healthy persons between the muscular development and the size and strength of the bones. In puny individuals with small and flabby muscles the skeleton is usually more or less under-developed. In this nutritional relationship between the muscles and the bones the muscles constitute the controlling factor. In a similar manner the bony walls of the cranium and thorax undergo changes corresponding to changes in the viscera which they contain.

Thorax.—In bed-ridden individuals and those suffering from wasting diseases the involution of the lungs from diminished functional activity or their diminution in size from pathological changes causes alterations in the shape and contour of the thorax, which tends to assume permanently the EXPIRATORY FORM; while an active life in the open air by increasing the volume of the lungs modifies the chest, which under these circumstances tends to assume permanently the INSPIRATORY FORM. Analogous changes in the chest result from lesions which increase the volume of the thoracic viscera, as in pulmonary emphysema and great cardiac enlargement. The point for the student to bear in mind is that many general and local changes in the form of the chest are primarily due to visceral disease and not to disease of the bones. Some of these are more marked when the

visceral disease takes place early in life, as in precordial prominence and the development of Harrison's furrows, others late in life, as in fibroid phthisis and emphysema. There are, however, exceptions to this general statement, an example of which is to be found in the changes of the shape of the chest which result from disease of the spine, as kyphosis.

Cranium.—The skull may be abnormal in size and shape either as the result of arrest of development of the brain with or without malformation, or as the result of pathological increase in the size of the brain. The short diameters and peculiar shape of the head of the microcephalic idiot and the globe-like cranium of chronic hydrocephalus developing at birth or in early infancy with its wide sutures, open fontanelles, and card-like thinness of the bones, are examples of the influence exerted by changes in the soft parts upon the bony walls containing them.

Skeletal Changes.—The bones themselves undergo pathological changes. These changes may be general or local. In ACROMEGALY there is hypertrophy of the bones of the hands, feet, and face, especially the inferior maxilla. The clavicles, sternum, and in some instances the long bones of the extremities also participate in the over-growth. In OSTEITIS DEFORMANS or PAGET'S DISEASE there is thickening of the bones of the skull and changes in those of the face, the outline of which becomes triangular with the apex at the chin; the long bones are involved and become deformed. In RICKETS, a disease of childhood, the head is large and square, the forehead prominent, the anterior fontanelle open, the epiphyses of the long bones are enlarged, nodules develop at the junction of the ribs with their cartilages. Changes in the shape of the chest and protrusion of the sternum cause the deformity known as CHICKEN OR PIGEON BREAST. The spine is curved, the clavicle bent, the pelvis deformed, and the long bones of the lower extremities show deformity. Rachitic children are often bow-legged; those who reach maturity are under-sized. OSTEOMALACIA is characterized by resorption of the lime salts. The bending of the softened bones under the action of gravity and muscular tension gives rise to extraordinary deformities. These affect the spine, thorax, pelvis, and long bones. In some instances the superficial bones crepitate upon pressure and can be indented by the finger. They are readily fractured and this accident may follow a trifling fall or blow or, in the case of the femur or humerus, result from the muscular force exerted in turning in bed. PULMONARY OSTEO-ARTHROPATHY—OSTÉO-ARTHROPATHIE HYPERTROPHIANTE PNEUMONIQUE OF MARIE—a condition encountered in certain chronic diseases of the lungs and pleura, is characterized by bulbous enlargement of the terminal phalanges of the fingers and toes and of the distal epiphyses of the bones of the upper and lower extremities. The finger-nails are hypertrophied and strongly incurved. The bones of the head and face are not affected.

JOINTS.

There are affections of the joints which lie on the border line between surgery and medicine. To the former belong traumatic and operative conditions; to the latter lesions arising in consequence of various constitutional affections. Commonly the question of diagnosis first rests with

the medical clinician. Those joint affections which properly come within the scope of internal medicine may be comprehensively described as the MEDICAL ARTHROPATHIES. The large and small joints may be affected. The chief symptoms are pain, especially upon movement, impairment of function, and the signs of inflammation or disorganization, namely, changes in color, size, and shape. The requisites to the proper examination of a diseased joint are a knowledge of the local anatomy and pathology and of the constitutional diseases in which joint affections occur.

Pain.—Pain is an important symptom. It may be spontaneous. More commonly it is caused by movement. Pain upon pressure occurs in acute forms of arthritis and is often intense. Pain is commonly referred to the affected joint, sometimes to a distant part, as the pain in the knee in hip-joint disease. In consequence of the freer movement permitted by muscular relaxation during sleep the pain is worse at night. There may be insomnia, or sleep may from time to time be broken by sudden agonizing pain. This is especially the case in tuberculous joint-disease. The patient very often awakes with a sharp cry of pain. The pain in myalgia and various forms of neuritis is frequently attributed to diseases of the joint; upon movement the pain is found not to involve the joint, but other structures, and the joint is neither tender nor swollen. In chronic joint affections movement is sometimes attended by a sensation of grating or crepitus, or there may be a catching sensation attended with crackling sometimes audible at a distance.

Color.—The color of the joint in acute inflammation is pinkish or red; when intense it is cyanotic or dusky. When there is marked periarticular œdema the overlying skin is pale.

Changes in Size.—In acute inflammation the joints are usually enlarged. This enlargement is attended with alteration in the contour. These changes are due to effusion, which may be articular or periarticular. The former may be serous, purulent, or hemorrhagic. The latter may be œdematous or exudative. These conditions are often combined. In chronic arthritis there is infiltration of the tissues entering into the formation of the joint. Enlargement due to effusion within the joint may be recognized by palpation, especially in large joints. In the knee the patella floats. Rounded local swellings fluctuating upon palpation may indicate the distention of the synovial sac. Enlargement may be due to changes in the ends of the bones.

Irregular diminution in the size may occur in chronic disease of the joints, as rheumatoid arthritis or other diseases characterized by resorption or retrogressive processes. Not only the tissues of the joint but the periarticular structures undergo atrophy and subluxations occur, or there may be diminution in the soft parts with thickening of the bones. All these processes are associated with changes in contour.

The posture is of importance. In forms of acute arthritis, flexion or semi-flexion and immobility are present—the attitude of least tension and therefore of least pain. The mobility of the joint is determined by passive movement. Fixation may be voluntary because it relieves pain. It may result from muscular spasm or large effusion. Sudden locking of a joint, especially the knee, may be due to floating cartilages or "joint mice"

becoming arrested between the anterior surface of the bones and the capsular ligament. In late cases the immobility is due to ankylosis, which may be adhesive, fibrous, or bony. Movement may be limited or prevented by the development of osteophytes in the region of the joints. Crepitus may be detected upon palpation.

Any of the joints may be involved in general diseases. The knee, hip, and shoulder are especially important, because of the frequency with which they are implicated, the disabling results, and the tendency to disorganization and ankylosis.

The medical arthropathies are inflammatory or infective, degenerative, and neuropathic.

Primary Arthritis.—Simple acute synovitis with effusion is very common especially in adolescents and young adults. It most frequently

FIG. 171.—Tophaceous deposits in gout.

involves the knee-joint. Traumatism and sudden chilling are causes. Some of the cases appear to be monarticular rheumatism with trifling fever. There is marked tendency to recurrence and chronicity.

Rheumatic Fever.—The affected joints are swollen, hot, usually slightly reddened, and painful upon motion. The amount of swelling is variable. The intra-articular effusion is usually slight or moderate, the periarticular œdema being commonly marked. When the wrists and ankles are implicated there is marked swelling of the hands and feet. The joint effusion of rheumatic fever is fugacious. The tendency to rapidly subside in one joint and develop in others is characteristic. The process is rarely limited to a single joint. Any joints may be affected, but the knees, ankles, and wrists are especially liable to the rheumatic inflammation.

Chronic Rheumatism.—This term is applied to a chronic condition in which the joints are painful, stiff, moderately swollen, and but slightly deformed. It is common in individuals who have been much exposed to the vicissitudes of the weather or have lived in damp places. Its etiological affinity to rheumatic fever may well be questioned. Some of the cases described under this term are undoubtedly subacute forms of rheumatoid

arthritis. In others the process is gouty. Cases of adhesive chronic arthritis have been described under the term chronic rheumatism. Very fat persons with small bones at or beyond middle life often suffer from painful knees. There is nothing to indicate gouty or rheumatic disease and no sign of actual inflammation. The pain is brought on by standing or walking and is often intense. There may be tenderness. The condition is mechanical, the bearing surface being inadequate to the weight of the body. Other articulations are not involved.

Gout.—This form of arthritis is due to the precipitation of salts of uric acid in the joint structures. The metatarsophalangeal joint of the great toe is first and most commonly affected, but other joints and especially the knee and ankle are occasionally involved. There is rapid swelling with heat, tension, and a bluish-red glistening skin.

Arthritis Deformans.—Implication of the joints is usually symmetrical though monarticular forms occur. First one or two joints only are involved. Gradually others are implicated and cases occur in which

FIG. 172.—Heberden's nodes (page 301, Vol. II).

all the joints suffer. Attacks of acute inflammation are succeeded by periods of quiescence, but after each attack the evidences of disintegration are more pronounced. The ligaments of the small joints, especially of the hands, are relaxed and the bones of the phalanges under the action of gravity very often form an obtuse angle with the metacarpal bones toward the ulnar side. Atrophic changes in the muscles and other structures relating to the affected joints occur in extreme cases. All the articulations may become ankylosed and the patient bedridden and almost completely helpless. There are partial or monarticular forms which occur in old persons. The spine may be involved—spondylitis deformans—with pain, anæsthesia, and muscular atrophy. In other cases the spine is involved together with the shoulder- and hip-joints and nervous symptoms are less prominent. Kyphosis and fixation occur.

Infective Arthritis.—Inflammatory joint affections frequently develop during convalescence from the acute infectious diseases. One or more joints show signs of inflammation. This form of arthritis is frequent after scarlet fever and sometimes occurs in cerebrospinal meningitis, the variolous diseases, dengue, and enteric fever. An

acute arthritis going on to suppuration with disorganization of the joint occurs in septic conditions. The joint affection which accompanies osteomyelitis is attended with high fever and constitutional disturbances.

Fig. 173.—Arthritis deformans.—Jefferson Hospital.

Gonorrhœal Arthritis. — Frequently one joint only is involved, sometimes several. The knee, wrist, and ankle frequently suffer. Tenosynovitis may occur. Fever is moderate or absent, or there is great pain on movement, and the joint affection is frequently persistent and disabling.

Arthritis in Hemorrhagic Diseases.—Acute arthritis, more or less intense and suggestive of the joint affection of rheumatic fever, occurs in forms of purpura and in hæmophilia. It is the larger joints that are chiefly affected. Intra-articular hemorrhage may occur. Arthritis is an occasional complication of scurvy.

Fig. 174.—Arthritis deformans with extreme ulnar deformity.—Jefferson Hospital.

Tuberculous Arthritis.—Tuberculous joint disease is common. It is often secondary to tuberculosis of the bones. It was formerly known as white swelling—*tumor albus*. The process is comparatively subacute but tends to permanent disorganization. Tuberculous joints are usually swollen. In the course of the disease chronic inflammatory infiltration takes place into the capsule, ligaments, and periarticular connective tissue. Caseation and softening result in abscess formation and burrowing along the lines of least resistance. Tortuous fistulous passages occur. The hip, elbow, knee, and wrist are frequently affected. There may be evidences of tuberculosis in the lungs or elsewhere. More commonly the process is limited to the affected joint and adjacent structures.

Syphilis.—The acute joint affection of new-born infants sometimes regarded as rheumatic is mostly syphilitic. It is a form of primary exudative arthritis with fibrous thickening of the capsule. Gummatous inflammation in the neighboring tissues may involve a joint by extension. In

acquired syphilis subacute synovitis occasionally occurs during the period of eruption. The sternoclavicular joint shows a peculiar liability. In late syphilis, forms of chronic arthritis, the result of gummatous infiltration of the tissues forming the joint, occur.

FIG. 175.—Gonorrhœal arthritis.—Pennsylvania Hospital.

Actinomycosis.—The joints are sometimes involved by metastasis. In other cases they are invaded by extension, as when the disease reaches the articulations of the cervical vertebræ or when prevertebral actinomycosis attacks the spine or the disease extends from the thorax to the sternoclavicular joints or from the abdomen to the hip-joints.

Neuropathic Joint Affections.—Hysteria especially may simulate disease of the joints. The impairment of function is caused by contracture of muscles. Pain is more diffuse and spontaneous than in actual arthritis. The patient avoids movement and does not coöperate in the examination. The signs of effusion, inflammation, and erosion are lacking. These are the cases in which spontaneous cures sometimes occur under profound mental suggestion. In some instances, from prolonged disuse, infiltration, and thickening of the periarticular tissues, false ankylosis and atrophy of the associated muscles occur. It is important to bear in mind that hysterical symptoms may be superadded to those of actual joint disease. The differential diagnosis between traumatic joint disease and a hysterical joint in traumatic hysteria is occasionally attended with difficulty. Vasomotor changes with swelling,

FIG. 176.—Ataxic knee-joint.—Young.

tension, and redness sometimes occur and the surface temperature may be two or three degrees higher than that in the axilla. These symptoms are not associated with fever or the evidences of constitutional disturbance and are commonly transitory and recurrent.

More important are the changes that take place in connection with certain diseases of the nervous system—CHARCOT'S JOINTS, TABETIC JOINTS—particularly locomotor ataxia, syringomyelia, less frequently in anterior poliomyelitis and other diseases of the spinal cord. The joint affection in tabes is much more common in the joints of the lower extremities, especially the knee, less frequent in the hip and ankle; that of syringomyelia is by far more common in the upper extremities. The derangements are primarily trophoneurotic. The process is frequently monarticular. The pathological and clinical changes correspond to those of the milder and graver forms of rheumatoid arthritis. In the more severe forms they differ in suddenness of onset, intra-articular effusion, and a rapid, disintegrating course without pain. Subluxations and luxations take place. When the tarsal articulations are implicated flat-foot occurs with characteristic deformities—the tabetic foot.

MUSCULATURE.

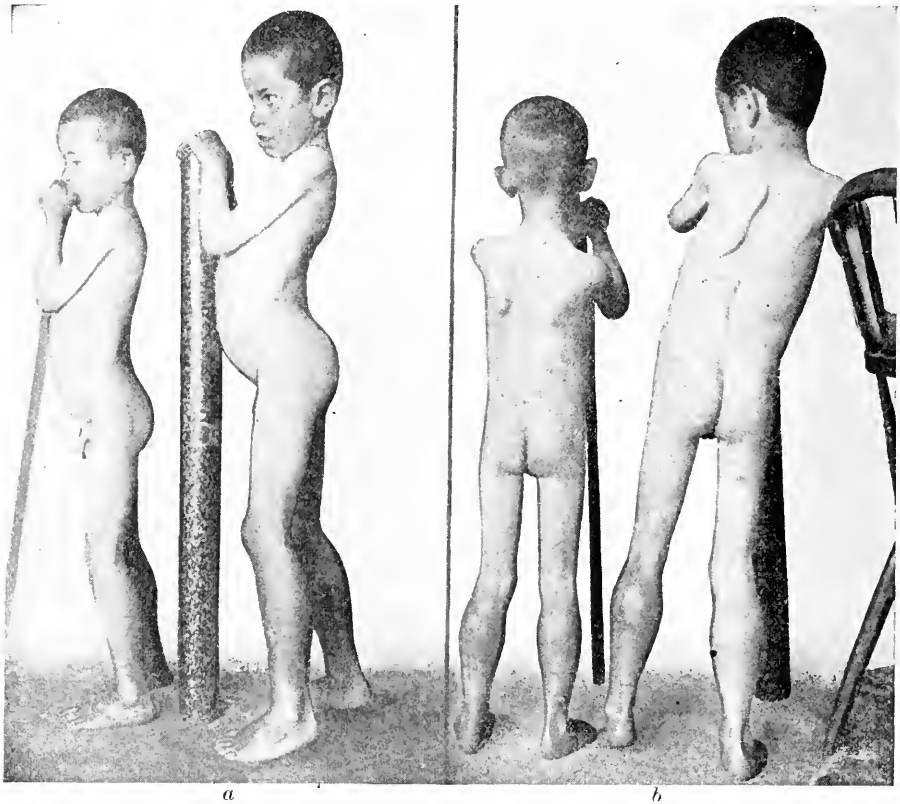
Diagnostic criteria of importance are obtained by an examination of the condition of the muscles. Wide variations in the bulk and tonicity of the general musculature is encountered within the limits of health. These variations depend largely upon the hereditary constitution, occupation, and bodily activities of the individual and are not of diagnostic significance. Trophic derangements result in hypertrophy and atrophy.

Hypertrophy.—True hypertrophy, that is to say, increased volume with increase of power, is exceedingly rare. It occurs in Thomsen's disease. CONGENITAL HYPERTROPHIA MUSCULORUM VERA has been described. Pathological increase in the muscles is almost always a pseudohypertrophy. The abnormal volume is not due to an increase in the contractile tissue but to a proliferation of the connective tissue and fat. This muscular dystrophy occurs in its most pronounced form in the so-called pseudohypertrophic muscular paralysis of childhood, and very rarely in some of the affected muscles in certain cases of chronic progressive muscular atrophy.

Atrophy.—Atrophy of the muscles may be simple or inactivity atrophy —the atrophy of disuse. The affected muscles are diminished in size, soft, and flaccid; there is loss of the contractile substance; the interstitial connective tissue is not increased. This form of atrophy occurs in certain forms of paralysis, and supervenes upon mechanical fixation of a limb or the prolonged immobility resulting from joint pain or ankylosis. Complete loss of movement usually gives rise to a high grade of simple atrophy. Atrophy from disuse rarely attains the degree often seen in the degenerative atrophies. In simple atrophy there is general diminution in the volume of the affected limb, while in the degenerative atrophies single muscles or groups of muscles are exclusively or chiefly involved. The electrical reactions in simple atrophy are quantitatively and not qualitatively changed. The nutritional muscular atrophy which occurs in starvation, in the course of acute infections, and in the chronic wasting diseases must be regarded as a diffuse form of simple atrophy.

Myoidema.—This phenomenon consists in a sudden contraction of muscular fibres when smartly tapped with the finger or hammer, with transitory humping at the point of impact. It is manifested in muscles that are undergoing rapid wasting, especially in phthisis, and is as a rule best developed in the muscles of the chest.

Degenerative Atrophy.—The degenerative muscular atrophies, which are characterized not only by loss of contractile substance but also



a *b*

FIG. 177.—Pseudohypertrophic muscular paralysis. Brothers, eight and ten years old. *a*, showing the lordosis; *b*, showing atrophy of back and enlarged calves.—Rotch.

by an overgrowth of the interstitial connective tissue, may be referred to two groups: (a) the progressive muscular atrophies, and (b) the atrophic paralyses.

The progressive muscular atrophies may be divided into myopathic, peripheral, and central or nuclear according to the seat of the essential pathological process, which may primarily involve the muscles, or result from an acute or chronic peripheral neuritis, or from degenerative changes in the ganglion cells of the anterior horns of the cord, or the motor nuclei of the brain. There is progressive atrophy of individual muscles and muscle groups; diffuse atrophy of an entire limb occurs only in advanced stages; the strength of the muscles is diminished in proportion to the diminution of their volume. In this respect the progressive muscular

atrophies are in contrast with the secondary degenerative atrophies which follow the atrophic paralyses. In the latter the paralysis comes first, the atrophy afterwards. The discrimination between myopathic, neural, and nuclear muscular atrophies rests upon the fact that in the different forms particular groups of muscles are affected. In the myopathic forms of degenerative atrophy—the muscular dystrophies—the following principal types occur: 1. Pseudohypertrophic muscular atrophy of childhood—

a b

FIG. 178.—*a*, infantile atrophy from improper feeding (female ten months old); *b*, recovery after three months.—Rotch.

the so-called pseudohypertrophic muscular paralysis. 2. The juvenile type of Erb—dystrophia musculorum progressiva; the atrophy begins in the shoulder girdle and is not preceded by pseudohypertrophy. 3. The juvenile type of Leyden-Möbius; the atrophy begins in the lower extremities. This form is closely allied to the progressive pseudohypertrophy of childhood. 4. The infantile type of Duchenne—the facio-scapulo-humeral

FIG. 179.—General atrophy of the muscles in a case of cerebrospinal fever; fifty-fifth day of illness.—Royer.

type of Landouzy-Dejerine. This form begins in the face. The loss of power in the muscles of expression gives rise to the characteristic FACIES MYOPATHICA. The eyes can no longer be completely closed, the cheeks are sunken, the lips thickened and everted, speech is impaired, and the ordinary changes in the countenance in laughter and crying are not seen. The myopathic atrophies are commonly hereditary and almost always show themselves in early life. Neural atrophy begins commonly in the under extremities in the distribution of the peroneal nerve—the peroneal type of Charcot and Marie—and may lead to the development of club-foot, usually pes equinus or pes equinovarus. It differs from other forms of

myopathic atrophy in the frequent occurrence of derangements of sensation, pain, and fibrillary contractions and in the occasional presence of the reaction of degeneration. In many cases of peripheral neuritis the affected muscles undergo degenerative atrophy. Spinal or nuclear atrophy usually first shows itself in the intrinsic muscles of the hand and by extension early involves the tongue, lips, palate, pharynx, and larynx, giving rise to the picture of bulbar paralysis. Fibrillary contractions of the muscles are common and reactions of degeneration occur. The disease develops almost exclusively in adult life and is not hereditary.

The Atrophic Paralyses.—The muscles undergo secondary degenerative atrophy. The lesion which interferes with the transmission of motor impulses at the same time interrupts trophic influences to the muscle. The paralysis shows itself first and is followed by atrophy, which in the course of some weeks becomes marked and often reaches a very high grade. The reactions are those of degeneration. In this form of degenerative atrophy fibrillary contractions are frequently present.

THE POSTURE, ATTITUDE, AND GAIT.

Posture.

Patients who are very ill of an acute disease or in the advanced stages of chronic disease are usually seen in bed; those suffering from trifling affections or in whom the symptoms of grave disease are not yet urgent or disabling continue to be about, but this is not always the case. Whether, on the one hand, a patient remains up and about, endeavoring to attend to his ordinary duties while suffering from serious symptoms or, on the other hand, betakes himself to bed upon the occurrence of trifling symptoms is often a matter of temperament. It is not uncommon for a patient suffering from enteric fever to come to the consultation room or dispensary in the second week of the attack with a temperature of 104° F. (40° C.) and a well-developed rose rash—WALKING TYPHOID. Patients who realize their condition very often feel compelled by circumstances to continue the discharge of a daily duty or are buoyed up by the hope of speedy improvement, and again there are acute diseases which run a favorable course which begin with urgent and distressing symptoms. The physician usually finds those patients in bed who have high fever, prostration, or a general sense of serious illness, and those who suffer from dyspnœa, pain, vertigo, and other symptoms intensified by movement or exertion. In meningitis, peritonitis, rheumatic fever, pericarditis, typical croupous pneumonia, and in well-developed cases of the acute exanthemata it is impossible for the patient to be out of bed. It is to be noted, however, that upon the appearance of the eruption in the variolous diseases the symptoms of onset often undergo such an amelioration that the patient regards himself as convalescent and insists upon getting out of bed.

Decubitus is the posture of the patient in bed. It is of diagnostic importance. It is in moderate illness, as in health, easy and unconstrained. The patient arranges the bed-clothes, changes his position when it has become uncomfortable, lies naturally upon his back—ACTIVE DORSAL

DECUBITUS—or turns upon the side—ACTIVE LATERAL DECUBITUS. The posture of weak, helpless, or unconscious individuals in bed is wholly different. The muscles play little part in maintaining the position. The relaxed body yields to the law of gravity and sinks toward the foot of the bed, where it remains. The patient, even when his breathing is hindered and his position is uncomfortable, is unable to change it. The attendants must again and again lift him upon the pillows. The condition is wholly passive—PASSIVE DORSAL DECUBITUS. In rare instances the patient in this state lies upon the side—PASSIVE LATERAL DECUBITUS.

Forced or imperative attitudes are very characteristic of certain diseases. The following are the most important:

The Dorsal Posture.—In acute peritonitis, whether general or local, the patient lies upon the back with the thighs flexed upon the abdomen and the legs upon the thighs. Movement is avoided and the patient shrinks from pressure upon the abdomen.

The Reclining Dorsal or the Sitting Posture.—In diseases attended with difficult respiration, especially certain diseases of the respiratory and circulatory organs and the kidneys, the patients are forced to assume a semi-upright posture on the bed-rest or propped up with pillows, or to sit upright. Attempts to lie flat in bed increase the difficulty of respiration. The sitting position relieves it by favoring the action of the accessory respiratory muscles, especially when the arms are used to elevate and fix the shoulders. In the case of peritoneal effusions the respiratory movement of the diaphragm is less interfered with in the sitting posture unless the effusion be very large, in which case the abdomen is somewhat compressed by the thighs. This attitude, furthermore, favors the return of the venous blood from the brain. For this reason high grades of dyspnœa are described under the term ORTHOPNŒA. When the difficulty of respiration is extreme the patients can no longer remain in bed but are obliged to sit upright, fixing the shoulders by placing the hands upon the side of the chair or its arms in order to facilitate the use of the accessory muscles and to relieve the abdomen from the pressure of the thighs. The distress is also to some extent relieved by the gravitation of venous blood and the fluid of general dropsical effusions to the lower extremities. Orthopnœa is present during the paroxysms of asthma, in extreme cases of valvular disease of the heart with ruptured compensation, in large pleural and pericardial effusions, in massive peritoneal effusions, and in general anasarca, which may be cardiac or renal but is very often cardiorenal. It occurs also in advanced pulmonary emphysema and in obstructive diseases of the larynx, as croup and diphtheria. Except in extreme cases it is usually paroxysmal, the attack being brought on by movement, coughing, conversation, or other exertion.

Lateral Postures.—Patients suffering with unilateral disease of the thoracic organs very often lie upon the affected side. This is especially the case in large pneumonic exudates, pleural and pericardial effusions, and other conditions which greatly diminish the respiratory surface of the affected lung. In this posture the respiratory excursus of the sound side is not hampered by the weight of the diseased organs. In painful conditions, however, the patients sometimes lie upon the sound side. In acute

fibrinous pleurisy the pain of which is greatly intensified by breathing, the lateral decubitus upon the affected side is assumed by preference because the weight of the body somewhat diminishes the respiratory excursus of that side of the chest. Patients suffering from heart disease and many individuals in good health lie more comfortably upon one side than upon the other; sometimes the right side is preferred, sometimes the left. In cardiac hypertrophy the patients usually lie more comfortably upon the left side, and in large aneurisms of the aorta, upon the affected side. Patients suffering from harassing cough in the dorsal position are sometimes relieved by turning upon one side. This happens in certain cases of unilateral pulmonary cavity and the relief is obtained by turning upon the affected side. The explanation of this phenomenon is purely physical; while the patient lies upon his back or upon the sound side the secretion formed in the cavity escapes into the bronchus little by little, causing irritation which manifests itself by cough, while, on the other hand, if he continues to lie upon the affected side it collects without producing reflex cough until the cavity overflows. The lateral decubitus with the thighs and legs flexed upon the abdomen and the spine and neck strongly arched forward is usually assumed during the pains of parturition and is common in hepatic and intestinal colic. In acute cerebrospinal meningitis the patient frequently lies upon the side with the thighs and legs strongly flexed and the spine extended in the position of OPISTHOTONOS. In some cases the lower extremities are extended—COMPLETE OPISTHOTONOS.

The ventral posture is sometimes assumed in cases of abdominal pain, as colic, gastralgia, or enteralgia. The patient lies prone upon the bed with his face buried in the pillow. Tenderness upon abdominal pressure, as in peritonitis, renders this attitude impossible. It sometimes affords relief to the pain of abdominal aneurism and in certain cases of caries of the spine. In most cases of gastric ulcer this posture is avoided on account of the epigastric tenderness upon pressure. In some cases of this disease, however, the pain is relieved by the ventral decubitus, probably because the ulcer is so situated as to escape in this position the pressure of the contents of the stomach. Patients suffering from headache very often assume this posture.

Restlessness in bed is a very common symptom. The patient is unable to maintain the same position for any length of time; he tosses about, turns from side to side, fusses at the bed-clothes, and his hands and feet are in constant motion. Restlessness may be the manifestation of nervous irritability or of pain. It is common in affections attended with burning and itching of the skin, as scarlet fever and urticaria. It occurs also in some cases of shock and accompanies profuse hemorrhage, in which case it is attended with pallor, urgent thirst, and rapid, small pulse. In truth the association of restlessness with these symptoms, occurring suddenly without visible bleeding, warrants a provisional diagnosis of internal hemorrhage. The term JACTITATION is used to designate a high degree of restlessness. The patient tosses about violently; the constant efforts of the attendants are necessary to keep him in bed. Jactitation occurs in maniacal delirium, in cases of violent chorea, in which it is accompanied by constant twitching of the muscles, as a temporary manifestation in

some forms of hysteria, and in a high degree during the stage of clonic convulsions in epilepsy.

Opisthotonos, predominating tonic contraction of the spinal muscles, so that the body rests upon the head and heels; emprosthotonos, or bending forward of the trunk; pleurothotonos, arched lateral posture;

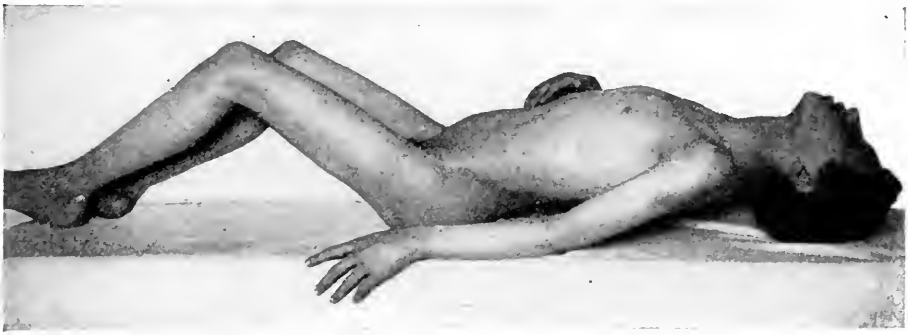


Fig. 180.—Opisthotonos in a case of epidemic cerebrospinal meningitis.—Royer.

Fig. 181.—Pleurothotonos in a case of epidemic cerebrospinal meningitis.—Royer.

and orthotonos, in which the trunk and neck are rigidly extended in a straight line, are all symptoms that occur in tetanus and in some cases of meningitis and strychnine poisoning.

Attitude.

The attitude and movements of patients who are able to be about frequently convey important information in regard to their condition. The young and the strong carry themselves erect and walk briskly and firmly; the aged and feeble and those mentally depressed are bowed and move slowly and with effort. The convalescent from a prostrating disease is at first weak and shaky; he can scarcely stand; an hour in the arm-chair fatigues him. In a little time he makes the journey around his room with

slow and uncertain gait, and is soon obliged to rest. With returning strength comes the erect carriage and firmer step. Modifications of attitude and gait constitute characteristic symptoms in many diseases. In general they are due to skeletal defects, as in caries of the spine, hip-joint disease, or ankylosis of the knee; derangements of the muscular power or function, as in pseudohypertrophic muscular paralysis, chorea, and the shaking palsies; derangements of the balance between antagonistic muscle groups, as in forms of spinal curvature and club-foot; derangements of coördination, as in cerebellar disease and tabes; forms of paralysis, as in hemiplegia, anterior poliomyelitis; and contractures, as in the cross-legged progression of children suffering from spastic paraplegia.

Station is technically the ability to maintain the erect position while standing. It depends largely upon muscular and visual coördination. Within limits it is better the wider the base of support, hence the test should be made with the feet parallel and the heels and toes touching, first with the eyes open, later with them closed. Hinsdale found in normal individuals of both sexes the average oscillation in the above position, as determined by an instrument devised for the purpose, to be about an inch in a forward and backward line and three-quarters of an inch laterally. The oscillation in children is greater than that in adults. Upon closing the eyes it is increased about 50 per cent. In diseases characterized by impairment of the power of coördination, as tabes and lesions of the cerebellum, station is greatly impaired and the patient may be wholly unable, under the conditions of the test and with closed eyes, to keep his balance—ROMBERG'S SYMPTOM. During paroxysms of Ménière's disease—aural vertigo—the power of standing in the erect posture is wholly lost. *Astasia* is a term employed to designate inability to stand, *abasia* the inability to walk, in the absence of paralysis. ASTASIA-ABASIA is a syndrome of hysteria in which the patient is unable to stand or walk but can usually creep about like a child, upon the hands and knees.

The following peculiarities of attitude are to be noted:

In HEMIPLEGIA and paralysis of one leg the patient supports himself almost entirely upon the sound leg. In CHRONIC SCIATICA the patient spares the affected limb both in walking and standing by fixation of the hip-joint, and in doing so develops a scoliosis, the spinal column showing a double curvature, the lower convex, the upper, which is compensatory, concave toward the affected side, the general inclination of the body being toward the sound side. In PARALYSIS AGITANS the attitude is characteristic. The head and upper part of the body are inclined forward, the elbows and knees being slightly flexed. The striking appearance of the patient is heightened by the expressionless countenance, the tremor, and the movements of the fingers and hands. In PSEUDOHYPERTROPHIC PARALYSIS the patient stands with his feet separated, the belly protruding, and the shoulders thrown back as the result of marked lordosis. In the sitting posture the curvature of the spine is corrected.

Gait.

In a number of diseases, especially those affecting the nervous system, the gait is much modified and its peculiarities often justify conclusions regarding both functional derangements and anatomical lesions. The following symptomatic gaits are frequently observed:

The Paraplegic Gait.—In paresis of the lower extremities the gait is feeble and uncertain. Both feet are slowly advanced and dragged upon the floor. The patient stumbles over trifling inequalities and elevations of the surface. The loss of power is frequently more marked on one side than on the other. Crutches become necessary and at length the loss of power is complete. This gait is seen in chronic myelitis.

The Hemiplegic Gait.—When the hemiplegic has sufficiently recovered to walk, the gait is characteristic. The sound limb is advanced, the paralyzed limb dragged after it. In other cases the step of the paralyzed limb is accomplished by lifting the pelvis and a movement of circumduction. When contractures have taken place the affected arm is rigid, strongly flexed at the elbow and wrist and carried across the body, and the fingers and thumb are flexed upon the palm.

The Spastic Gait.—In spastic paresis of the lower extremities such as occurs in forms of spinal paralysis there is peculiar stiffness of the legs, which are scarcely bent at the hip- and knee-joints, while the thighs interfere with each other by reason of the contraction of the adductors. The contraction of the gastrocnemii produces pes equinus. The patient walks with two canes and in stepping leans upon one, lifting the pelvis of the opposite side as he steps, and dragging the foot in circumduction. In some cases the contact of the foot with the floor produces ankle clonus which adds to the peculiarity of the gait. A modification of the spastic gait, sometimes seen in children, is known as cross-legged progression. In consequence of the contraction of the adductors and calf muscles there is close circumduction of the knees, and in stepping the legs are crossed and the advancing foot brought down not only in front of but to the outside of its fellow.

The Steppage Gait.—In some cases of peripheral neuritis the paralysis of the extensors of the feet causes a peculiar modification in progression. In stepping forward the knee is strongly flexed and the foot sharply advanced in order that the dragging toes may be lifted from the ground; the heel is brought down first and the appearance is that of a person stepping over obstructions.

The waddling gait occurs in pseudohypertrophic muscular paralysis and is not less characteristic than the attitude in this disease. In consequence of the lordosis the shoulders are thrust back and the belly forward, the legs are separated, the feet raised slowly with the toes dropping, the centre of gravity being alternately shifted over the foot upon which the patient throws his weight. The manner in which the child, after lying down upon the floor, gets up is especially characteristic. He rolls over upon the abdomen, gets upon all fours, and first extends the arms, then the legs. The hands are next drawn toward the legs until he can grasp one knee with the corresponding hand. He pushes himself up until

the other knee can be grasped and assumes the erect posture by gradually raising the point of support of the hand upon the thigh. Late in the disease, when the atrophy involves the muscles of the upper extremities, it becomes impossible to rise.

The ataxic gait is that of incoördination of the lower extremities. It is observed in its most typical form in tabes dorsalis. In stepping the foot is raised higher than usual with a jerk and rapidly advanced with an awkward and irregular movement, the toes slightly drooping. It is then brought down with an abrupt stamp upon the heel or the entire sole. Progression is irregular and it is impossible for the patient to walk with one foot before the other, as in following a crack upon the floor or a chalked line. He walks with a swaying motion. The legs are separated in order to increase the base of support, which is further enlarged as the disease makes progress by the use first of one cane, later of two. In advanced cases walking becomes impossible without the aid of one or even two attendants. Finally, the power of locomotion is entirely lost. These symptoms of impaired coördination are greatly increased upon closing the eyes. Patients who can go about fairly well in daylight cannot walk at all in the dark.

The gait of sciatica derives its characteristics from muscular fixation of the hip-joint voluntarily brought about to diminish pain.

The Gait in Chorea.—In severe chorea the irregular muscular contractions interfere greatly with ordinary movements. The gait of the patient is often hopping or sliding, sometimes it resembles the movements of skating. In the worst cases walking becomes impossible.

The reeling or staggering gait is a form of the ataxic gait. It occurs in conditions attended with marked disturbance of coördination, such as drunkenness, cerebellar disease, lesions of the labyrinth, and some forms of paralysis of the muscles of the eye.

The Festinating Gait.—This modification of walking occurs in paralysis agitans and is not less characteristic than the attitude in that disease. The patient bends forward, the elbows are slightly abducted and flexed, the knees are also flexed, and the patient walks with the appearance of haste, as though to overtake his advancing centre of gravity. He cannot halt at once. The peculiarity of the gait is largely due to stiffness and weakness of the muscles. The gait is sometimes described as propulsive. A similar gait and inability to stop immediately sometimes shows itself in exhausted pedestrians. Retropulsion may occur.

Posture and Movements of Infants.

The position and movements of infants are of diagnostic importance. The healthy baby uses its muscles and joints. Its postures are active, its movements constant, and a source of evident pleasure. It loves to be fondled and played with. How different the baby who is really ill! Its postures are passive. Its head drops and rolls from side to side with the motion of the pillow upon which it rests. Its limbs dangle helplessly, and voluntary movements are slight and infrequent. In many febrile diseases there is cerebral irritation, shown by the drawn face and head pressure deep into the pillow. In severe rickets there is tenderness

of the muscles and bones, motion is painful and therefore avoided; in infantile scurvy a similar condition exists, and in well-developed cases the attitude is almost diagnostic, the child lying upon its back with the thighs and legs strongly flexed, shunning all movements and screaming with fear if it is approached. In cerebrospinal fever and other forms of meningitis there is painful retraction of the muscles of the back of the neck—opisthotonos.

IV.

TEMPERATURE; FEVER; HYPOTHERMIA; SIGNIFICANCE OF ABNORMAL TEMPERATURES.

TEMPERATURE.

Variations in the temperature of the body constitute symptoms of great importance both in acute and in chronic disease. From the earliest times practitioners estimated the heat of the body by the hand and thus sought to determine the presence or absence of fever. The introduction of the clinical thermometer into medical practice marked an important advance in modern medicine. (See Part II, Clinical Thermometry.)

Heat Mechanism.—The temperature of homothermous or warm-blooded animals is constant within narrow limits and is not materially influenced by changes in the temperature of the medium in which the organism lives. In the human being the amount of heat produced and dissipated at different parts of the body varies. The equilibrium of temperature is maintained in part by direct conduction but chiefly by the circulating blood and lymph. The internal parts of the body have nevertheless a higher temperature than the external and some internal organs are warmer than others. The heat production is greater in organs when they are active than when they are at rest, and the temperature varies in different regions of the surface of the body. The heat mechanism is made up of two factors: (a) heat production or *thermogenesis*, and (b) heat dissipation or *thermolysis*. Under normal conditions these two functions so nearly balance that the mean bodily temperature is maintained within very narrow limits. The regulating mechanism is expressed by the term *thermotaxis*. It is obvious that thermotaxis may be deranged by alterations in either thermogenesis or thermolysis.

Thermogenesis accompanies oxidation. Hence almost every structure of the body may be regarded as the source of heat. In this respect the skeletal muscles and the glands play the chief part. The general thermogenic centres have been shown to be in the spinal cord. Thermogenic centres probably exist in the caudate nuclei, pons, and medulla oblongata; excitation of these regions is followed by a rise in heat production—puncture pyrexia. They are therefore known as thermo-accelerator centres. Irritation of the region of the sulcus cruciatus and at the junction of the supra-Sylvian and post-Sylvian fissures in the dog is followed by a decrease in heat production. These centres are therefore known as thermo-inhibitory.

Thermolysis or heat dissipation is the result of radiation and conduction from the surface, of the evaporation of water from the lungs and skin, and of the warming of the food, drink, and inspired air.

Thermotaxis or heat regulation is brought about by reciprocal changes in heat production and heat dissipation through the action of cutaneous impulses and of variations in the temperature of the blood upon the thermogenic and thermolytic centres. Thus in an animal exposed to moderate cold, heat dissipation is increased, but cutaneous impulses are generated which excite the thermogenic centres and heat production also is increased, whereas an increase of the temperature of the blood increases the activity of the thermolytic process. In either case the temperature of the body is maintained. Under abnormal conditions this reciprocal influence is deranged.

Abnormal thermotaxis is a term used to designate the regulation of the heat mechanism under pathological conditions in which the body temperature is maintained at a range higher or lower than that of health.

Under ordinary circumstances the presence or absence of hyperthermia may be determined by the hand, but this mode of observation yields no accurate data either for comparison or record. An impression as to the surface temperature is thus obtained but this does not always correspond with the internal temperature of the body. During a chill the temperature of the skin, in consequence of the contraction of the arterioles, is in most instances greatly reduced, while the internal temperature, as determined by the thermometer, is high. On the other hand, when the skin is active and perspiring and evaporation is prevented by the bed-clothing, the surface may feel hot to the hand while the internal temperature remains normal. The normal axillary temperature ranges about 98.6° F.—37° C. It undergoes diurnal oscillations of a degree to a degree and a half, falling to 97.5°–98° F. in the early hours of the morning and rising to 99°–99.3° F. toward evening. It is very probable that this physiological oscillation is dependent upon the alternations of sleep and waking. Observations upon men who have habitually slept during the day and watched during the night have shown an inversion of the curve. A slight physiological rise takes place during gastric digestion. Violent physical exercise is frequently followed by a temporary rise of two or three degrees; this fact may, in part at least, explain the elevation of temperature sometimes observed after a violent general convulsion and which is very common in the *status epilepticus*. In children and adolescents the range is somewhat higher than in adults and also less stable, that is to say, the diurnal physiological oscillations are slightly greater and the sensitiveness of the temperature to pathogenic influences more marked.

Kieffer states that careful observation has shown that permanent increase of external heat in the tropics is followed by a rise of bodily temperature of .05° F. for every degree of external heat above the mean annual norm and that as a direct consequence the respiratory function is diminished, the pulse-frequency slightly decreased, the digestion, appetite, and assimilation unfavorably affected, the functional activity of the skin greatly increased, and the nervous system distinctly depressed.

In aged persons the diurnal temperature range in health is slightly lower and may fall to 97° F. (36.1° C.). In very aged persons, on the other hand, the range may be as high as in children.

The action of prolonged or intense heat and cold upon the temperature must be regarded as pathological.

Abnormal Temperature.—Variations in the body temperature may be plus to progressively higher ranges, designated subfebrile and febrile, the latter comprising (a) slight fever, (b) moderate fever, (c) high fever, (d) hyperpyrexia; or minus—subnormal temperature and the temperature of collapse.

The term pyrexia is used to designate conditions characterized by elevation of temperature; hyperpyrexia, those marked by excessively high temperature; and apyrexia, the absence of fever. Hypothermia is the term applied to conditions in which the temperature is subnormal.

FEVER.

Elevation of temperature alone does not constitute fever. Extreme transient rises of 104° F. (40° C.) have been observed after violent, prolonged gymnastic exercises, and much higher temperatures in hysteria, in neither case associated with the other symptoms which enter into the modern conception of fever. These symptoms are, in addition to elevation of temperature, subjective sensations of illness, cerebral phenomena, weakness, loss of appetite, thirst, increased frequency of pulse and respiration, altered urine, and derangements in nutrition which cause wasting of the body. It is furthermore essential to our conception of fever and necessary to the complete manifestation of the symptom-complex that the process should occupy a certain time. There are, however, febrile periods of minimal duration, as for example in the course of the ague paroxysm, in which most of the objective symptoms occur or in which, if the paroxysm is repeated for some time at quotidian or tertian intervals, all of them, including wasting of the body, are manifest. On the other hand, the acute febrile infectious diseases usually run a self-limited course, measured by days or weeks; again, in certain of the chronic infections, as forms of tuberculosis, there may be fever every day for months. Nor are these symptoms altogether dependent upon or caused by the elevation of temperature, as is shown by the fact that artificial over-heating of the body produces certain of them but not all, that in different diseases their intensity by no means corresponds to the degree of the temperature, and that marked falls of temperature can occur either spontaneously or as the result of antipyretic treatment without a corresponding amelioration in other respects. Elevation of temperature is nevertheless a constant and essential element in the condition known as fever and in certain cases dominates the clinical picture. In a majority of instances, however, the associated conditions constitute a much more important measure of the gravity of the case than the range of temperature.

Causes of Fever.—It is evident that the causes of fever act through the nervous system and thus produce derangements of the heat-regulating function. At the same time they also produce derangements of the normal

tissue changes with increased oxidation and heat production. They consist of soluble toxic substances circulating in the blood and are, (a) the result of infection by micro-organisms, which may be general or local, or (b) the result of intoxication, which may arise within the body itself from faulty metabolism or be introduced from without, as in food poisoning. In cases of infection with profound nutritive disturbances toxins derived from both these sources are present. In either event, whether the fever-producing agent be a toxin produced by the growth and development of micro-organisms or an albumose, ferment, or ptomaine produced by faulty cell metamorphosis within the organs or tissues themselves, the condition constitutes a toxæmia.

Sapræmia is an infection of the blood by putrefactive products.

It is probable that in the rare cases of fever attributed to intense emotion, as fright, or to violent pain or peripheral irritation, the rise of temperature is caused by the sudden derangement of physiological processes, with the production of toxins, rather than by direct action upon the heat-regulating processes, and that in many, though not all, of the cases of cerebral disease accompanied by fever, as thrombosis, hemorrhage, and tumor, the elevation of temperature is due to local infection rather than to implication of the heat centres, while the symptom-complex and the condition of the blood in sunstroke render it highly probable that the elevation of temperature is due not so much to the direct effect of heat upon the nervous system as to toxic substances generated by the action of heat upon the tissues of the body and especially upon the muscles. It is thus seen that many different pathogenic principles developed within the body or introduced from without are directly or indirectly capable of producing the reaction which we designate by the term fever.

Symptoms of Fever.—These substances not only cause elevation of temperature and more or less marked disturbances of nutrition but they also produce subjective sensations of illness and cerebral symptoms, such as headache, somnolence, stupor, and, in grave cases, coma and delirium, which may be mild and wandering or active and maniacal. Among the effects produced upon the nervous system must be included the profound sensation of weakness often present in the early stages of febrile diseases and which bears no direct relation to the inability to take food or to the wasting of the tissues of the body which occurs later. They produce derangements of the normal secretions, which are manifested on the part of the skin by dryness and heat or, in some cases, and especially at the time of defervescence, by profuse, even colliquative sweating, on the part of the gastro-intestinal tract by thirst, loss of appetite, dry, furred tongue, impaired digestion, and constipation, and on the part of the urinary apparatus by scanty, high-colored urine of increased specific gravity.

Pulse in Fever.—Derangement of the pulse-frequency is a constant phenomenon of fever. To what extent it is due to elevation of the temperature and to what extent to the action of fever-producing toxins upon the nervous system cannot be determined. In almost all cases of fever there is an acceleration of the pulse-rate, the frequency of which usually corresponds to the intensity of the fever. Liebermeister found that for every degree centigrade (1.8° F.) of elevation of temperature above the

normal there is an increase of eight beats of the pulse. This parallelism between the temperature and pulse may be regarded as relatively favorable, whereas a greatly increased pulse-frequency indicates serious cardiac or vasomotor disturbance and is of unfavorable prognostic significance. A pulse-rate of 140–160 in the adult while resting quietly in bed is in itself a very serious symptom. The pulse-frequency in children suffering from febrile diseases is relatively high. In phthisis with moderate fever or even in the absence of fever there is commonly a quickened pulse. There are cases in which, notwithstanding marked elevation of temperature, the pulse-rate remains low. This departure from the ordinary parallelism is of diagnostic importance. High temperatures with slow pulse are observed in cases of cerebral disease in which there is pressure at the base, as tuberculous meningitis, in yellow fever, and in febrile diseases in individuals suffering from cardiac lesions attended by diminished pulse-frequency, as sclerosis of the coronary arteries and myocarditis. It is to some extent characteristic of enteric fever that the pulse-frequency is moderate as compared with the elevation of temperature, and this want of correspondence is of importance in the differential diagnosis between enteric fever and acute miliary tuberculosis or septicæmia, in both of which the pulse-rate is high.

Respiration in Fever.—Increased frequency of respiration occurs in almost all cases of fever. That this phenomenon is in part due to the stimulating effect of the heated blood upon the respiratory centre has been shown experimentally; exposure to artificial heat increases the frequency of breathing. That it is also in part due to the direct action of the fever-producing toxins upon the respiratory centre is rendered probable by the fact that the acceleration of breathing bears no direct ratio to the elevation of temperature but varies greatly at the same temperature in different diseases. It is a matter of experience that cases of febrile disease in which, in the absence of complications on the part of the respiratory organs, the respiration frequency is greatly increased are almost always of grave import.

Emaciation.—Wasting accompanies fever. Even in febrile attacks of moderate duration the loss of flesh may be marked; in prolonged fevers emaciation may be extreme. The blood undergoes analogous changes, the patient becomes anæmic, and the loss of flesh at the close of a prolonged fever is not more striking than the pallor. A decrease in the number of the erythrocytes accompanies all cases of pyrexia, but requires some time to become manifest. There is progressive loss of the albumins of the plasma.

Pyrexia a Symptom.—The clinical significance of fever would be much less important were it not for the fact that the febrile movement, in its mode of onset, intensity, course, and decline, bears a relation to the particular morbid condition in which it occurs, frequently definite and always suggestive.

Until recently much stress was laid upon the distinction between symptomatic fever and essential or idiopathic fever. The former was regarded as a manifestation of some local malady, the latter as constituting the actual disease. The stimulus given to the study of causes by the science of bacteriology has shown that this distinction is more apparent than real and that in the light of modern pathology pyrexia is always a symptom

Idiopathic Fever.—Nevertheless there is a group of acute infectious diseases in which fever is not only constantly present but also the most conspicuous symptom, and in which the morbid process is literally coextensive with the febrile movement, which is self-limited, the illness beginning with the rise of temperature and the convalescence setting in with defervescence. This group constitutes the idiopathic fevers or, more simply, the fevers.

Varieties.—Subdivisions, arranged according to the course of the febrile movement, are (a) the continued fevers, as influenza and enteric fever, and (b) the periodical (malarial) fevers, as intermittent, remittent, and pernicious fever. In some of the continued fevers other symptoms, as eruptions, are no less constant or characteristic than the course of the fever,—a fact which led to the establishment of a further subdivision upon an entirely different basis of classification, which comprises the exanthemata or the eruptive fevers. Furthermore, in certain of the diseases which are regarded as continued fevers, a characteristic periodicity occurs, or the course of the disease is interrupted by periods of apyrexia of considerable duration, an example of which is relapsing fever, whereas in the periodical fevers, strictly so-called, namely, the malarial infections, there are certain cases in which the febrile movement lacks distinct periodicity—continued malarial fever—or is absent altogether—malarial infection without fever. On the other hand there is a large group of diseases that has nothing to do with malaria in which the occurrence of febrile paroxysms, separated by very definite periods of apyrexia, in other words, distinct periodicity, is characteristic—for example, the hectic fever of pulmonary tuberculosis, hepatic fever, urethral fever, and the fever in some cases of malignant endocarditis. Finally, there are local and general infections in which the symptom fever is inconstant and irregular. For these and other reasons, the principal of which is that fever is always symptomatic and never of itself an actual disease, the distinction between symptomatic fever and essential or idiopathic fever has been abandoned—a long step in the direction of a scientific or etiological basis for the classification of diseases. Terms and phrases that have long lost their original significance remain to encumber the literature and embarrass the study of medicine and the period is remote when we shall cease to speak of scarlet fever or yellow fever.

Type in Fever.—Type is a term loosely used to indicate the intensity of fever. Thus we speak of fever of mild type or fever of grave type. It is applied more accurately to the course or range of the temperature as depicted upon clinical charts. There are three principal types of fever: (a) the continued, in which the limits of the diurnal range do not usually exceed 1.8° F. (1° C.), the fall occurring in the morning, the rise in the evening. This is about the measure of the diurnal oscillation in health. There is, therefore, a parallelism between the temperature of health and fever of the continued type, the latter being elevated two or more degrees above the former and fluctuating in harmony with it. Since the temperature range upon the chart is represented not by a straight but by a curved line showing the diurnal oscillations it is better to describe this as the subcontinuous type. Fever of this type is characteristic of the fastigium

of uncomplicated enteric fever. (b) The REMITTENT type, characterized by falls of several degrees in the temperature, which does not, however, reach the normal. The remissions may take place at any hour of the day and are often accompanied by free sweating. They are followed in the course of a few hours by exacerbations of greater or less extent. There is no parallelism between fever of this type and the normal temperature range. This is the type seen in some forms of estivo-autumnal malaria and in septic conditions. (c) The INTERMITTENT type, characterized by a fall of temperature

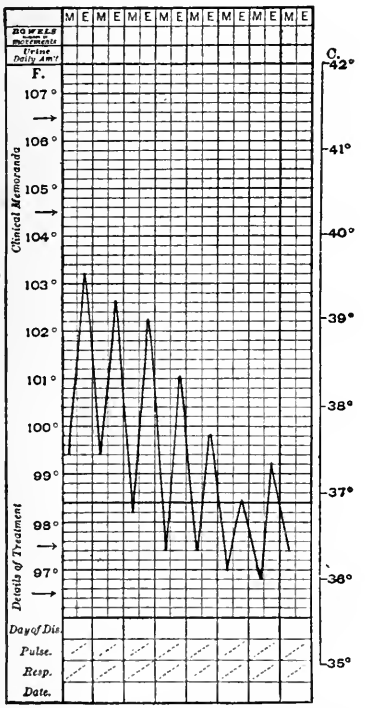


FIG. 182.—Fever of remittent type: lysis in enteric fever.

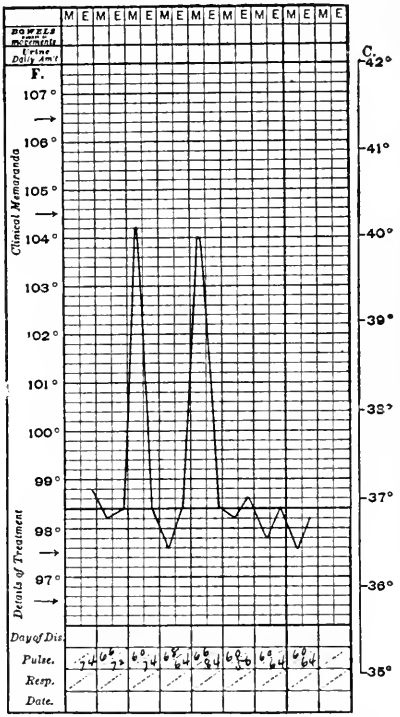


FIG. 183.—Intermittent malarial fever, tertian type; single tertian infection. Man, aged thirty-three.

from febrile ranges to the normal or below it, a period of apyrexia of variable duration, and the recurrence of fever. The febrile paroxysms are of short duration as compared with the intermission and commonly begin with a chill and terminate in profuse sweating. During the intermission the patient usually feels fairly comfortable or quite well. Fever of this type occurs in malaria. The repetition of the paroxysms may extend over a considerable time. Intermittent fever in which the paroxysm recurs daily is known as *quotidian;* when the paroxysm recurs upon the third day, including the day of onset, it is *tertian;* when it recurs upon the fourth day, *quartan.* The paroxysms may occur at any period of the day and usually at the same hour. In malaria they ordinarily recur in the forenoon, in hectic fever in the afternoon. (d) THE INVERSE TYPE. The tem-

perature in fever of the continued type and in many cases of the remittent type undergoes diurnal oscillations of wider excursus than those of health but corresponding to them in time. That is, the remission occurs in the early morning hours, the exacerbation toward evening. In exceptional cases the remission takes place in the evening and the exacerbation in the morning—*inverse type*. Fever of this type occasionally occurs in tuberculosis and in rare instances in enteric fever.

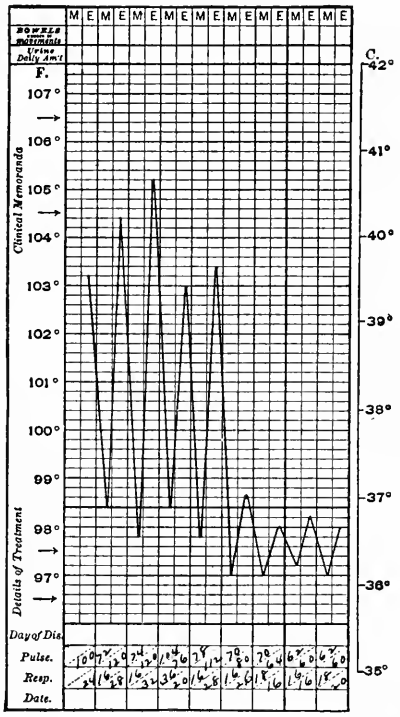


Fig. 184.—Intermittent malarial fever, quotidian type; double tertian infection. Man, aged twenty-nine.

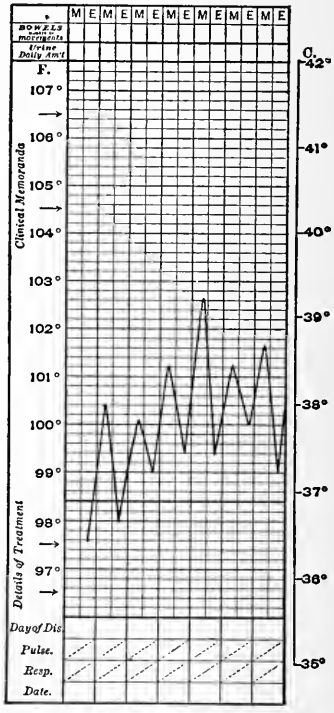


Fig. 185.—Temperature of inverse type.

Atypical Fever.—In many febrile diseases the temperature range is altogether irregular. This is especially the case in diseases in which the symptoms in general are irregular or atypical, as diphtheria and the various septic infections.

The Type of Fever in Particular Diseases.—Many of the febrile infections have a characteristic temperature range. The febrile movement in uncomplicated cases on the one hand is self-limited and on the other undergoes definite modifications at different stages in the course of the affection and upon the occurrence of special manifestations, as the appearance of an eruption. In a more narrow sense the temperature range in such diseases is said to be typical or to conform to type. It is to be borne in mind, however, that marked departures from type may occur in consequence of variations in the intensity of the infection, peculiarities on the part of the

individual, the occurrence of complications, and from the action of drugs. The type of fever, both as regards the daily range and the temperature curve throughout the course of the attack, constitutes a valuable aid to diagnosis, and is always to be taken into consideration. It is rarely possible, however, to make a diagnosis from the temperature alone, nor is it desirable. In connection with the temperature we must consider the other symptoms and signs, the surrounding circumstances, and the previous treatment.

In well-developed cases, unmodified by complication or treatment, the temperature curve may be said to be characteristic in the following diseases: tertian and quartan malaria, enteric fever, typhus fever, relapsing fever, and croupous pneumonia. It conforms in a general way to type, but less closely, in scarlatina, measles, erysipelas, and the variolous diseases. It is variable and atypical in cerebrospinal fever, rheumatic fever, endocarditis, and the septic infections.

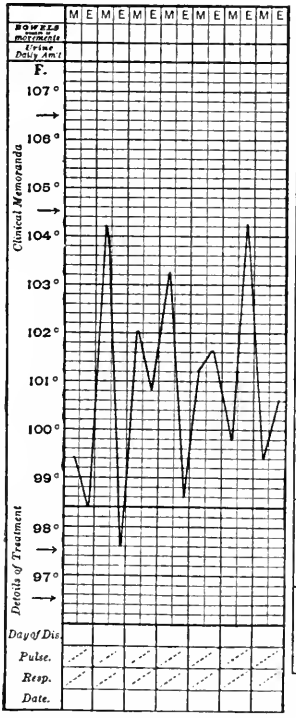


FIG. 186.—Fever of irregular periodicity.

Stages.—The course of the attack may be divided into (a) the stage of prodromes, (b) the onset or stage of invasion, (c) the fastigium, and (d) the defervescence or stage of decline. In typical cases of the different febrile diseases each of these periods has a definite duration and a characteristic curve upon the temperature chart.

(a) **The Stage of Prodromes.** — This period is usually marked by vague feelings of discomfort, lassitude, pain in the back, unsound sleep, and feverishness, the temperature reaching subfebrile or even mild febrile elevations in the later part of the day. These symptoms are often absent. Prodromes usually occur in diseases of gradual development. They are common in enteric fever.

(b) **The Onset or Stage of Invasion.**—The rise of temperature may be gradual or abrupt. When gradual the evening exacerbations exceed the morning remissions in such a way that the temperature rises progressively to the fastigium or acme. Under these circumstances the stage of invasion may occupy a period of several days, as in enteric fever. When abrupt the acme is reached at once or in the course of a few hours, as in scarlet fever, influenza, or croupous pneumonia. The onset is very often attended by chilliness or a chill. This symptom may vary in intensity from transient sensations of cold, with shivering, pallor, and slight cyanosis of the lips and finger-tips, to a severe and prolonged chill or rigor, with violent shaking or tremor of the whole body, chattering teeth, cold extremities, and marked cyanosis. The temperature of the surface of the body is much reduced and the patient experiences a sensation of extreme cold, whereas the internal temperature, taken in the rectum, is high, 104°–107° F. (40°–42° C.). The

violence of the chill commonly corresponds with the abruptness of the onset. The insidious and gradual invasion of enteric fever is not often attended by chills. The abrupt onset of croupous pneumonia very frequently manifests itself by a prolonged chill of great severity, occurring without warning in a condition of apparent health. The chill which ushers in the febrile paroxysm or ague fit of malaria is intense and prolonged, and the congestive chill of the algid variety of pernicious estivo-autumnal malaria may terminate in death. Chills occurring later in the attack may mark the development of an intercurrent disease, as croupous pneumonia in the course of enteric fever. The chills of malignant endocarditis cannot be distinguished from the ague paroxysm, the resemblance to which is frequently heightened by a regular periodicity. Ague-like chills occur in some cases of phthisis and are common in local suppurations with pent-up pus, cholelithiasis, and septic and other conditions attended by fever of intermittent type.

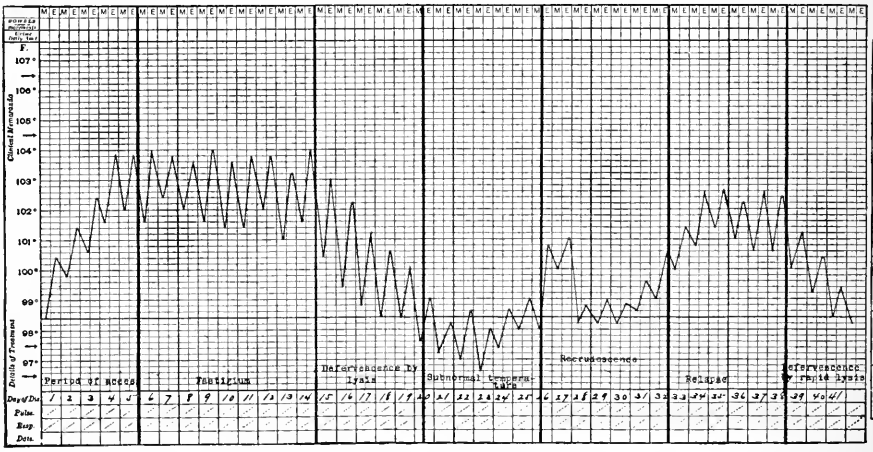


FIG. 187.—Temperature curve in enteric fever, with recrudescence and relapse.

The perturbation of the nervous system, which is manifested in the adult as a chill, may show itself in the child as a convulsion, sudden stupor, or very rarely as an outbreak of delirium. In the adult the onset of an acute febrile infection may be marked by sudden maniacal delirium and patients developing pneumonia or enteric fever have in some instances been regarded as insane and committed to an asylum.

The chill which attends the general or local infections and which is of varying intensity must be distinguished from the so-called nervous chill which sometimes occurs in persons of neurotic constitution under conditions of excitement, intense pain, moderate shock, or great fatigue. Under such circumstances there may be trembling and agitation, but the pulse remains good, the normal color is preserved, and the thermometer does not show a rise in temperature.

(c) **The Fastigium or Acme.**—Fastigium is literally the summit or ridge of a building. The temperature range in the continued fevers shows diurnal remissions and exacerbations corresponding to those of health, but somewhat greater. The elevation above the normal differs in different

diseases and in different cases of the same disease. In croupous pneumonia and in typhus and relapsing fevers the elevation is high. In many cases of enteric fever it is moderate. A parallelism with the temperature of health is to some extent maintained in the continued fevers. This parallelism may, however, be interrupted by accidents, as hemorrhage or perforation in enteric fever, complications, as empyema in pneumonia, the occurrence of pseudocrises, the action of antipyretic drugs, or the external application of cold by means of baths or otherwise. In the periodical fevers the

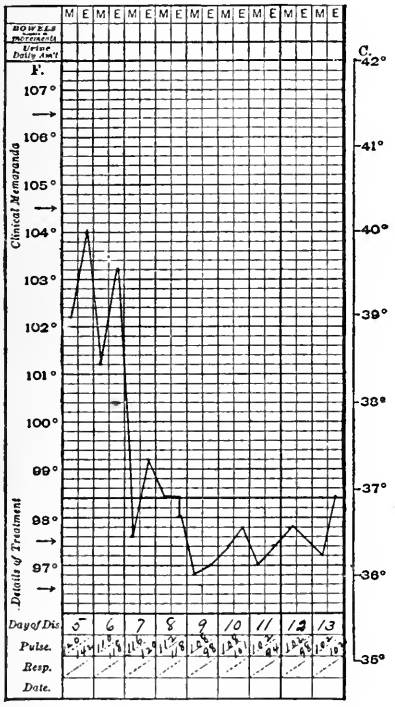


Fig. 188.—Croupous pneumonia in a child three years old. Defervescence by crisis on the evening of the sixth day.

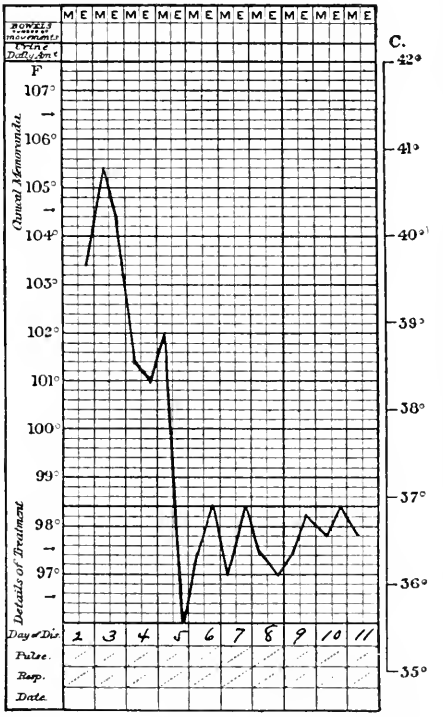


Fig. 189.—Pneumonia. Early defervescence; interrupted crisis.

diurnal range is much greater than in health. The term acme is used to indicate the summit of the range when the febrile movement is transient, as in influenza or the ague paroxysm.

An abundant hemorrhage from any surface, especially intestinal hemorrhage in enteric fever, causes an abrupt fall of the temperature to normal or below it. The shock of perforation is likewise accompanied by a fall of several degrees.

A normal temperature in the course of fever may thus assume the significance of an abnormal temperature.

In abortive cases of enteric fever, especially in children, the defervescence is often critical. Intercurrent diseases and complications may cause a rise above the range of the fastigium.

(d) **The Defervescence or Stage of Decline.**—The fall of temperature may be abrupt, or gradual. The former is known as *crisis* or *critical defervescence*, the latter as *lysis*. The abrupt fall in crisis amounts to several degrees in the course of a few hours. The temperature usually reaches subnormal ranges from which it reacts gradually. The fall may be broken by a slight rise—*interrupted crisis*. It is often attended by critical discharges, such as copious perspiration, passage of a large quantity of urine, or large liquid stools. Not infrequently it occurs during, or is followed by, a deep and prolonged sleep from which the patient awakes refreshed but weak and exhausted. There is a corresponding fall in the pulse and respiration frequency. The gradual fall in lysis takes place by progressive increase in the morning remissions and decrease in the evening exacerbations until normal or subnormal ranges are attained. This process frequently extends over several days, as in enteric fever. The term *rapid lysis* is applied to a gradual defervescence of shorter duration. Febrile diseases of sudden onset, such as croupous pneumonia, for instance, not infrequently terminate by crisis, while those of gradual invasion commonly terminate in lysis.

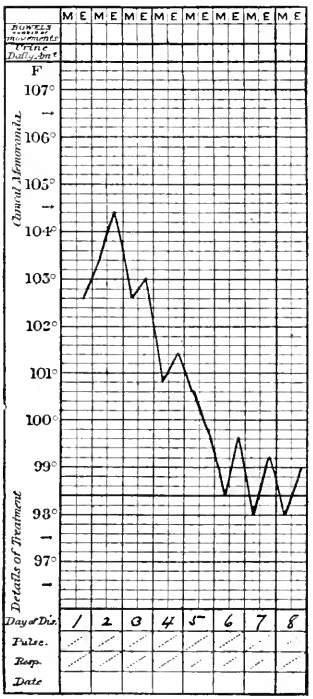


FIG. 190.—Scarlet fever; defervescence by lysis. Boy, aged twelve.

Persistence of fever beyond the normal period in a self-limited disease is due usually to a complication; sometimes to relapse. The febrile course of measles is frequently prolonged by bronchopneumonia; of scarlet fever by middle-ear disease, endo- or pericarditis, pleurisy, or nephritis; of enteric fever by phlebitis, abscess formation, cholecystitis, necrosis of cartilage or bone, some form of secondary infection, or by relapse. Cases of enteric fever extending to the fifth week or longer, in which no complication can be discovered, are mostly instances of intercurrent relapse.

The Temperature during Convalescence.—In the early days of convalescence from acute febrile disease the temperature range is frequently subnormal. It is also *labile*, that is to say, very readily disturbed by trifling influences, such as constipation, the return to solid food, mental excitement, or over-exertion. A transient rise of temperature produced by any of these causes is known as a *recrudescence*.

Relapse.—A recurrence of fever, together with the characteristic symptoms of the primary attack, due to reinfection. Instances of two or more relapses—*multiple relapse*—are of occasional occurrence. That form of relapse which begins before the defervescence from the primary attack is completed is known as *intercurrent relapse*.

Bed Fever.—Patients who have passed through febrile diseases sometimes develop during convalescence a moderate febrile movement, the evening exacerbations ranging as high as 100° or 101° F. This fever tends to run on indefinitely but may quickly disappear if the patient is allowed to sit up. A diagnosis of bed fever should never be made until other fever-producing conditions are excluded.

Paroxysmal Fever.—The fever recurs at intervals. The temperature is high and the accompanying symptoms usually severe. The febrile movement is of short duration and commonly preceded by a chill and followed by profuse sweating.

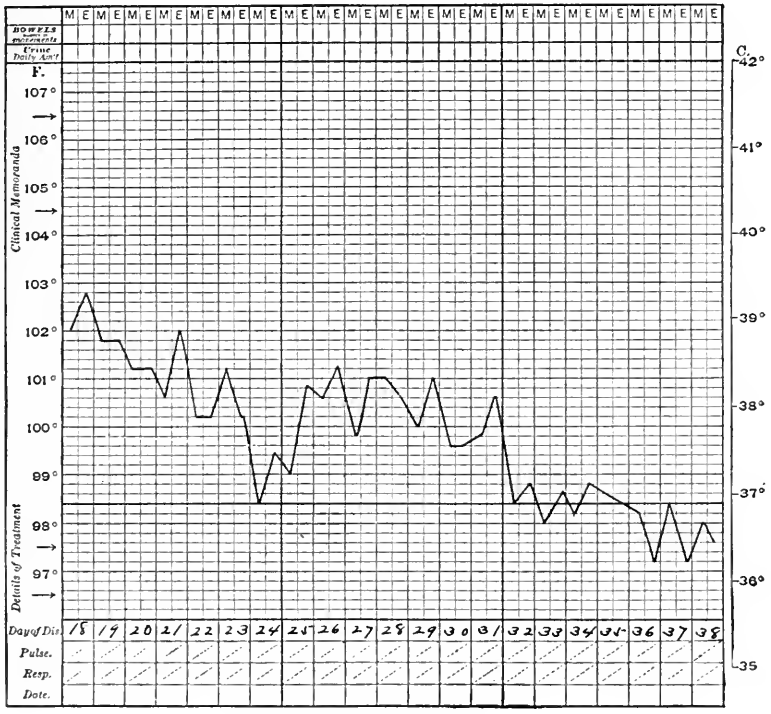


Fig. 191.—Interrupted lysis.

Periodical Fever.—The periodicity may be regular or irregular. Intermittent and remittent fevers are periodical. Tertian and quartan malaria constitute the very type of the regularly recurring periodical fevers. The periodicity of the estivo-autumnal fevers is not so well defined; the type is blurred and in some cases wholly obliterated. We observe forms of continued malarial fever due to estivo-autumnal infection.

Other febrile diseases are characterized by periodicity—a matter of great practical importance in diagnosis.

Paroxysmal fever, often of regular periodicity, may occur in the following conditions: (a) Abscess formation and other suppurative processes, as empyema. In cerebral abscess the temperature may be continuously normal or subnormal. Evacuation of pus and free drainage is fol-

lowed by disappearance of fever. (b) Pyæmia and septicæmia. (c) Malignant endocarditis. (d) Suppurative and infectious processes in the liver and bile-passages—*hepatic fever.* Under this heading are abscess of the liver, diffuse cholangitis, cholecystitis, inflammation of the hepatic, cystic, and common ducts, gall-stone disease, especially impacted gall-stones, and hypertrophic cirrhosis. (e) Infections of the genito-urinary tract, as cystitis and pyelitis, prostatic abscess, and after the passage of the catheter or sound—*catheter fever, urinary fever.* (f) Tuberculosis. Paroxysmal fever is present in the acute miliary form, the early stages of many cases and

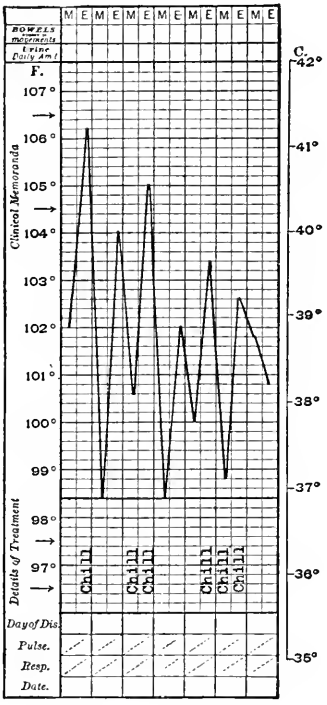


FIG. 192.—Urethral fever. Man, aged sixty-four.

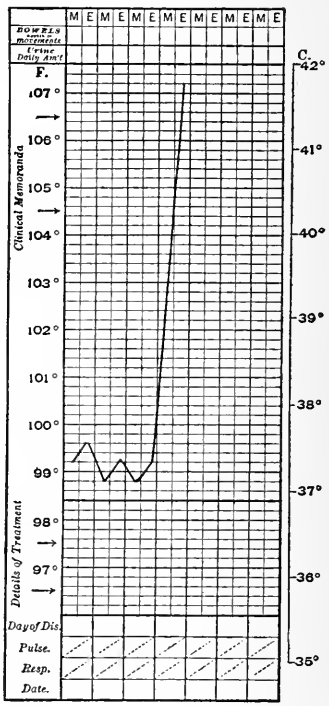


FIG. 193.—Cerebral hemorrhage. Hyperpyrexia. Pre-agonistic rise of temperature.

the later stages—after cavity formation—of almost all cases of pulmonary phthisis, and very often in acute tuberculous processes involving the bones, joints, and glands. Sometimes in the last few days of consumption the fever wholly ceases. (g) Hodgkin's disease and leukæmia. Periods of fever may be separated by prolonged periods of apyrexia. (h) Syphilis. The initial fever may come on within four or six weeks after infection and persist for several weeks. Paroxysmal fever in some instances accompanies the development of gummata and other lesions of the tertiary period. (i) Rapidly growing malignant neoplasms. (j) Very rarely in morphinism, the febrile paroxysm being preceded by a chill and followed by copious sweating. If the minimal temperatures fall to or below the normal the fever is intermittent in type; if they fail to reach the normal

it is of remittent type. In the course of any of the foregoing diseases the fever may change from one to the other of these types and frequently it becomes irregular and wholly atypical. In the course of defervescence by lysis as the fever gradually falls to normal it passes from the subcontinuous type of the fastigium first to the remittent type, then to intermittent.

The febrile paroxysms, in some cases of pyæmia, malignant endocarditis, and tuberculosis, and of disease of the liver and bile-passages, are ushered in by rigor and terminate by sweating, and recur with such regularity that they closely resemble the ague paroxysms of malaria. Errors of diagnosis are common, but readily avoided by close observation, examination of the blood, and the therapeutic test of quinine.

Hyperpyrexia.—Excessively high temperatures are occasionally observed. The thermometer may register 105.8° F. (41° C.) and higher in injuries involving the cervical portion of the spinal cord, and in tetanus, rheumatic fever, scarlet fever, enteric fever, yellow fever, and sunstroke. Very high temperatures occur in croupous pneumonia, the paroxysms of malarial fever, relapsing fever, and erysipelas. A marked rise may occur in the acute infections just before death—*pre-agonistic rise.* Excessive temperature when transient is not necessarily of grave prognostic import; if continued for some hours it is apt to be followed by death. Da Costa has recorded a temperature of 110° F. (43.3° C.) in a case of cerebral rheumatism, Jacobi has seen in scarlet fever 107.6° F. (42° C.), Sahli 113° F. (45° C.) in enteric fever, Richet 107° F. (41.7° C.) in sunstroke, with recovery. The literature contains many instances of recovery after such temperatures. There are well authenticated cases of even higher temperatures with recovery. The most remarkable is that of Teale, reported to the London Clinical Society in 1875. A lady fell from her horse and sustained serious spinal injuries. For sixty days she had frequent rises of temperature to 111.2° F. (44° C.) and higher but eventually recovered. Bryant, Guy's Hospital Reports, 1894, has recorded the facts of one hundred cases of hyperpyrexia, several of which, however, are not above suspicion. Many of the cases of excessively high temperature have occurred in hysterical persons and several of the most remarkable instances on record are obviously the result of deception.

HYPOTHERMIA.

Subnormal Temperature.—Hypothermia may be present under the following conditions:

(a) The intense action of external cold. A transient body temperature of 86° F. (30° C.) may occur, yet recovery take place.

(b) After a pronounced crisis at the close of an acute infectious disease, as pneumonia. Postcritical falls to 95° F. (35° C.) or even to 93.2° F. (34° C.) have been observed.

(c) In shock and collapse. The fall of temperature is associated with signs of failure of the circulation, frequent, small, or imperceptible pulse, colliquative sweating, great relaxation, and extreme pallor. The mind, except in the presence of cerebral lesions, usually remains clear. The condition may be transient or it may be the immediate forerunner of death.

Subnormal temperature may be the result of internal or external hemorrhage, traumatism, surgical operation, prolonged anæsthesia, the apoplectic insult in cerebral hemorrhage, embolism or thrombosis, the sudden rupture of a hollow viscus with the discharge of its contents into the peritoneum, or finally the action of intense pain or a sudden, overwhelming, depressing emotion in a neurotic individual. When reaction takes place the temperature rises very often to febrile ranges, either as the result of infection or, in the case of cerebral or spinal lesions, from irritation of the tissues which constitute

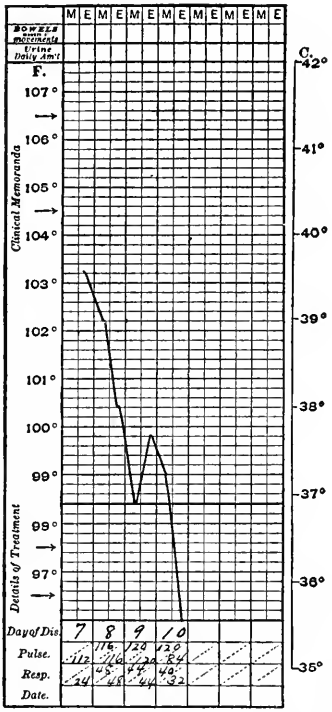


Fig. 194.—Pneumonia. Death in collapse after crisis.

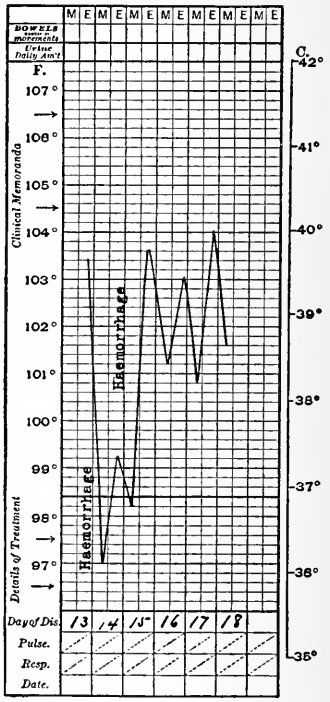


Fig. 195.—Enteric fever; subnormal temperature due to repeated hemorrhage.

the heat centres. In lesions of the cerebrospinal axis the reactive fever is frequently due to inflammatory reaction in the neighborhood of the lesion.

(d) In various conditions attended by greatly diminished tissue change or profound disturbance of the heat mechanism, as in the coma attending acute poisoning from alcohol, illuminating gas, carbolic acid, and other toxic agents, starvation, carcinoma of the œsophagus, other internal cancers, abscess of the brain, myxœdema, sclerema neonatorum, and in some forms of mental disease, as melancholia. Subnormal temperature ranges are also occasionally observed in profound anæmia, the terminal stages of tuberculous processes, especially tuberculous peritonitis, and in diabetes.

The Action of Drugs upon the Temperature.—Antipyretic drugs, most of which are synthetic products of coal-tar, while capable of produc-

ing marked effects upon febrile temperatures, have little influence upon the body temperature in health. Their free use in fever is followed by a tendency to collapse, and the resulting fall of temperature is of short duration.

External Antipyretics.—Cold baths or gradually cooled baths, sponging, packs, ice-bags, circulating coils for the application of iced water, and cold enemata reduce the febrile temperature not only without the perturbating effects of drugs but, if rightly employed, with a favorable influence upon the general condition of the patient.

Trifling rises of temperature follow the administration of full doses of atropine, cocaine, strychnine, caffeine, and certain other drugs, while correspondingly slight falls occur after morphine, quinine, alcohol, and the general anæsthetics.

THE SIGNIFICANCE OF ABNORMAL TEMPERATURES.

To recapitulate: A rise of temperature, if moderate, may be physiological—digestion, violent muscular effort. Such rises are commonly transient. If the rise be accompanied by other symptoms of fever it may indicate (a) an infection, either general or local; (b) an intoxication, which may arise within the body from faulty metabolism or be introduced from outside the body, as in the case of food or drink; (c) a lesion involving the heat-regulating mechanism of the nervous system.

As a rule there are associated symptoms which render practicable the differential diagnosis of these conditions.

A fall of temperature may indicate blood loss, which may be internal and concealed, as in a small rupture of the wall of the heart not presently fatal; a similar leakage from an aneurism; collapse, as in apoplexy; excessive radiation, as in exposure; diminished metabolism, as in convalescence, starvation, forms of poisoning, and certain nutritional and nervous diseases. The fall may be transient or sustained.

Whether the temperature be higher or lower than the normal it serves to exclude malingering and, as a rule, hysteria. It is important to bear in mind that remarkable departures from the normal temperature are observed in some cases of hysteria, and that the clever malingerer often plays tricks with the thermometer that are as difficult of detection as they are puzzling.

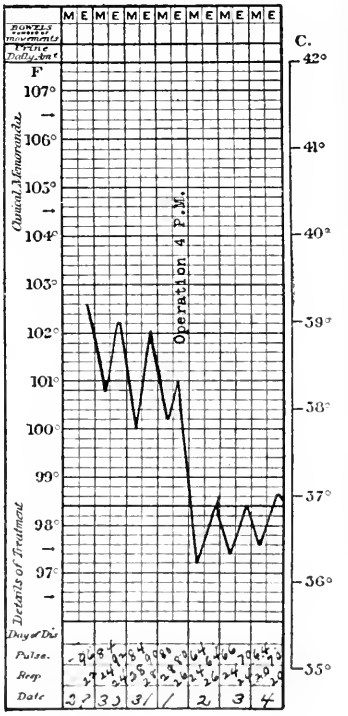


FIG. 196.—Ischiorectal abscess. Fever immediately relieved by incision and drainage.

The Prognostic Significance of Abnormal Temperature.—The height of the temperature is important, since the danger increases with the inten-

sity of the fever. A rise of 7.2° F. (4° C.) or 9° F. (5° C.) is of itself ominous. If sustained for some hours death almost always follows, though remarkable exceptions to this rule have been observed. Abrupt rises from the range of health, such as are seen in malaria or relapsing fever, are less dangerous than sudden rises above the fastigium in the course of enteric or other continued fever.

Variations from type in the temperature curve modify the prognosis unfavorably. The irregular pneumonias of moderate temperature, 102° F. (38.8° C.) are attended by greater danger to life than the typical forms with intense fever, 104° F. (40° C.) or higher.

The temperature in childhood is less stable than in adult life. It is elevated by slight causes, and reacts more readily to antipyretic treatment. Forms of ephemeral fever are more common than in later life. High temperatures are less dangerous.

V.

RESPIRATION; MODIFIED RESPIRATORY MOVEMENTS; COUGH AND ALLIED PHENOMENA; SIGNIFICANCE OF COUGH IN DIAGNOSIS; EXPECTORATION.

RESPIRATION.

The normal respiration-frequency in the adult is from 16 to 24 in the minute. The average pulse-frequency varies between 64 and 96. The normal pulse-respiration frequency is therefore 1 to 4–4.5. In early life the respiration is more rapid, the average being in the new-born 44; in the fifth year 26. Posture exerts a marked influence. In normal adults the average frequency while recumbent is 14, while sitting 20, and in the erect posture 22. These differences are exaggerated in those who are enfeebled by disease. The respiration is slightly less frequent in the morning than at night, and about one-fourth less during sleep. It is more rapid after eating and especially after a hearty meal, a fact which finds its explanation in the more limited excursus of the diaphragm when the stomach is full. The rate is very little influenced by the external temperature. It is modified by the internal temperature and much increased in fever. It increases with muscular activity. The respiration frequency may be modified within limits by an effort of the will and is profoundly affected by the emotions. Clinically the rate is often quickened by the knowledge on the part of the patient that his breaths are being counted. It is therefore important, if possible, to count without his being aware of it, for instance, when you appear to be counting the pulse. Failures of observation may be controlled by counting for one or more entire minutes. The rate and depth of the respirations bear an inverse relation to each other: the greater the frequency the less the depth, or the slower they are, the deeper. General abnormal conditions and local diseases, especially those which involve the organs of respiration, modify both the frequency and extent of the respiratory movements.

Tidal Air.—This term is used to designate the inflow and outflow of air during quiet respiration. It amounts to about 500 cubic centimetres—30 cubic inches. COMPLEMENTAL AIR is the volume that can be inspired after the completion of an ordinary inspiration; RESERVE OR SUPPLEMENTAL AIR, the volume that can be expelled after an ordinary expiration; RESIDUAL AIR, the volume remaining in the lungs after the most forcible expiration; STATIONARY AIR, the volume remaining in the lungs after an ordinary expiration and equal to the reserve air plus the residual air. VITAL CAPACITY is the volume of air that can be expired after the fullest inspiration. The average is about 3400 cubic centimetres for men and 2500 cubic centimetres for women. LUNG CAPACITY is the total quantity of air in the lungs after full inspiration, and is equal to the vital capacity plus the residual air.

Vital Capacity.—The measurement of the vital capacity is determined by various modifications of the spirometer devised by Hutchinson. It is affected by age, sex, stature, posture, occupation, and disease. It increases with age, the maximum being attained at about the thirty-fifth year. It is greater in men than in women of the same height in the ratio of 10 to 7.5. It increases in proportion to the stature up to the twenty-fifth year, and Arnold found that, in the adult, for each centimetre of increase or decrease of height beyond a mean standard there is a corresponding rise or fall in the vital capacity of 60 cubic centimetres for men and 40 for women. The ratio as modified by posture is 0.96 in the prone, 1.11 in the sitting or erect, and 1.13 in the standing position. The vital capacity is greater in those leading an active than in those who lead a sedentary life. It is obvious that improper clothing and tight lacing and all pathological conditions which interfere with the full and free expansion of the chest, whether general, as in wasting diseases of every kind, or local, as in thoracic or abdominal diseases, must diminish the vital capacity; nor is it to be overlooked that pregnancy or a sedentary life not in itself incompatible with excellent health may exert a similar influence. The spirometer, partly for these reasons, partly by reason of its inconvenience of application and uncertainty as an instrument of precision, and, finally, because there are other methods far more available and accurate, has fallen wholly into disuse in ordinary clinical work.

Peculiarities in the respiratory phenomena are expressed by the following terms:

EUPNŒA is a condition of normal respiration observed during bodily and mental quiet. APNŒA is a temporary suspension of the respiratory movements. HYPERPNŒA is a term used to designate increased respiratory activity. HEAT-DYSPNŒA and POLYPNŒA are forms of hyperpnœa due to direct excitation of the respiratory centres, as the result of an increase in the temperature of the blood or of reflex excitation of the cutaneous nerves by external heat. DYSPNŒA is difficult or labored breathing; the respiratory frequency is often less than normal but may be increased. ASPHYXIA OR SUFFOCATION is the term used to express the condition caused by deprivation of air. The respirations are at first increased in frequency and depth, then a period of increasing dyspnœa follows, with violent spasmodic expirations and convulsions. The final condition is that of collapse, which is ushered in by progressive slowness and shallowness of the respi-

rations, dilatation of the pupils, disappearance of the motor reflexes, loss of consciousness, convulsive twitching, and relaxation of the sphincters. The heart commonly continues to beat for some minutes after the cessation of breathing, so that by means of artificial respiration the patient may be restored to life. After death the blood is dark, the veins and lungs engorged, and the arteries empty.

Type in Respiration.—The filling of the lungs with air is brought to pass in part by the outward and upward movement of the ribs and sternum and in part by the contraction of the diaphragm. Either of these factors may predominate; hence a costal type of respiration and a diaphragmatic or abdominal type. In women the costal type is more pronounced; in men the abdominal. In the new-born the type is abdominal, in older children costal.

The type undergoes modifications in consequence of various pathological conditions which affect the costal or abdominal respiration.

Limitation of costal respiratory movements is caused by intrathoracic disease or by changes in the wall of the thorax. Dense pleural thickening, pulmonary consolidation, loss of pulmonary elasticity, effusions, large aneurisms, and tumors of every kind limit the respiratory excursus in the region involved. If one-sided, as is mostly the case, the unaffected lung takes upon itself additional work,—*vicarious respiration*,—and the increased respiratory movement of the sound side is in strong contrast to the restricted movement of the affected side. Calcification of the costal cartilages and the ankylosis of the costosternal articulations, which takes place in arthritis deformans and emphysema, interfere with the movements of the ribs and may convert the costal type of respiration in the female or the costo-abdominal in the male into the purely abdominal type.

Limitation of abdominal respiration may arise as the result of mechanical interference with movements of the diaphragm, paralysis of the diaphragm itself through flattening of its vault by the presence of pleural effusions, or in emphysema, and instinctively to avoid pain. The costal type may therefore be intensified and the diaphragmatic diminished in the following conditions: mechanically in all forms of marked distention of the abdomen, as advanced pregnancy, tympany, tumors, and ascites; acute inflammations of the serous membranes in relation with the diaphragm, as pleurisy, pericarditis, and peritonitis—the limitation of movement being in part the result of pain, in part the result of paresis of the musculature of the diaphragm; paralytic states involving the diaphragm, as multiple neuritis or progressive muscular atrophy.

MODIFIED RESPIRATORY MOVEMENTS.

Derangements of the Frequency and Rhythm of the Respiration.—(a) **Diminution in the Respiration Frequency—Oligopnœa.**—This symptom is common in stuporous conditions and in coma. It occurs in severe brain disorders, as hemorrhage, tumors, meningitis, in uræmia, diabetic coma, intense infections, and many forms of narcotic poisoning. As dissolution approaches the respiration declines in frequency. In all of these conditions the rhythm of the respiration may be deranged. The changes are due to altered function of the respiratory centre.

(b) **Increased Frequency—Polypnœa.**—This results from increased demands upon the respiratory function and constitutes an important element in the symptom-complex dyspnœa. It occurs also as a nervous symptom in hysteria and certain forms of cerebral disease.

(c) **Characteristic Derangements.**—1. Meningeal Respiration, Biot Breathing.—This form of breathing, as its name indicates, is common in meningitis, but may occur in other affections of the brain and in severe general infections and toxic conditions. It is characterized by pauses in breathing, which last from a few seconds to half a minute or longer and recur at regular or irregular periods. It is of unfavorable prognostic import. The characteristics of Biot's respiration are: Periods of apnœa, which vary in length and occur at irregular intervals; constant irregularity in rhythm, and in the force of the individual respirations; the frequent occurrence of deep sighs; uniformity of the expiratory level.

2. Cheyne-Stokes Respiration.—There are similar pauses in this form of breathing. They do not, however, occur as mere interruptions of respiration but are preceded by a gradual diminution in the depth and frequency of the respiratory acts until breathing wholly ceases. After a pause of several or many seconds the breathing is re-established. It is at first shallow and slow, but progressively increases and becomes by degrees more rapid, deeper, and sometimes urgent, until a maximum is attained. Then follows another gradual decrease, to be again followed in time by total arrest of respiratory movement. Cheyne-Stokes respiration is characterized by an exquisite periodicity. It is encountered in grave general conditions due to affections of the brain, heart, or respiratory organs, especially in individuals who have marked arteriosclerosis. It occurs also in uræmia and usually but by no means invariably in unconsciousness. This form of breathing may arise while consciousness is retained, and especially is this the case in chronic affections of the circulatory and respiratory organs. Under some circumstances consciousness is partially or wholly lost during the respiratory pauses and regained in the intervals of breathing. During the pauses there is sometimes a marked slowing of the pulse, with altered tension, and contraction of the pupils, but these phenomena bear no constant relation to the respiratory changes. During the acceleration in breathing which follows the pause the patients in some instances experience a desire for air and in other instances the sensation of having been roused from sleep. Cyanosis may occur during the pause. In certain cases Cheyne-Stokes respiration occurs only in sleep. Full doses of morphine are followed by an intensification of the phenomena, and Cheyne-Stokes respiration may first appear in the sleep which follows the admin

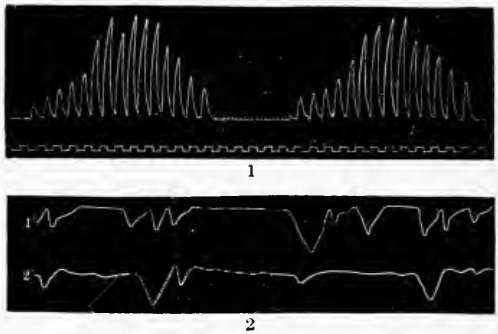


Fig. 197.—1. Cheyne-Stokes respiration pneumatography. 2. Biot's tracings illustrating the meningitic type of breathing. Down stroke = inspiration. (Am. Jour. Med. Science, March, 1911.)

istration of this drug. The prognosis of the underlying condition is always grave, and this form of respiration is seen in profound illness from which, exceptionally, the patient rallies in a short time or, and this is the general rule, presently dies. In some cases of cardiac or renal disease, however, Cheyne-Stokes respiration recurs from time to time for months.

3. Jerking Respiration.—The act may be spasmodic. Usually it is the inspiration that is jerking, less commonly the expiration, rarely both. Jerking inspiration is seen in sobbing, hysteria, hydrophobia, sometimes in asthma; jerking expiration, in acutely painful respiration, such as occurs in pleurisy, especially diaphragmatic, in pleurodynia, or in the case of a broken rib. Jerking respiration is more apt to occur when the breathing is of the costal type than when it is abdominal.

Dyspnœa.—This term includes a variety of respiratory derangements which, much as they may differ among themselves in detail, have one thing in common, namely, difficulty in breathing. The clinical conception, whatever the cause or whatever the derangement, rests upon inadequate oxygenation of the blood. Dyspnœa may arise from a deficiency of oxygen or from an excess of carbon dioxide in the blood. Cardiac and hemorrhagic dyspnœas are due chiefly to a deficient supply of oxygen. In cardiac dyspnœa the poor supply of blood to the tissues results from the enfeebled action of the heart. In hemorrhagic dyspnœa there is enfeebled action of the heart on the one hand and a diminished quantity of blood on the other. All conditions which lessen the force of the circulation or the quantity of hæmoglobin tend to cause dyspnœa; hence individuals who suffer from cardiac lesions or forms of anæmia, or who are enfeebled by disease, experience difficulty in respiration after slight exertion. Conditions which interfere with the interchange of oxygen and carbon dioxide in the lungs, such as bronchitis of the smaller tubes, forms of pneumonia, emphysema, extensive pulmonary tuberculosis, laryngeal obstruction, abdominal tumors, and large ascites, tend to the production of dyspnœa, especially upon exertion.

The respiration may be more or less frequent than normal. Two principal forms may be distinguished, namely, that in which the breathing is rapid and shallow and that in which it is slow and deep. In the former the ratio between inspiration and expiration is not usually much deranged; difficulty in breathing attends both acts and the condition is spoken of as *mixed dyspnœa.* In the latter the ratio is disordered, sometimes the inspiration, sometimes the expiration being more prolonged. To the one is applied the term *inspiratory dyspnœa;* to the other *expiratory dyspnœa.* The first is characterized by inspiratory, the second by expiratory stridor. In all forms of dyspnœa certain muscles which ordinarily are little or not at all used in respiration and have other functions are brought into play. They are the auxiliary muscles of respiration. Among them are the scaleni, trapezii, levatores scapulæ, the sternocleidomastoid, sterno- and thyrohyoid muscles, and the pectorales. The action of these muscles is more powerfully exerted in the erect or sitting posture—*orthopnœa*—the position usually assumed in inspiratory dyspnœa. In expiratory dyspnœa the abdominal muscles are used as auxiliary muscles.

In dyspnœa of high grade the muscles of facial expression are also brought into play, with the effect of dilating the nostrils and separating

the lips and jaws. The action of these muscles gives to the facies a very characteristic and distressed appearance. Very striking is the play of the nostrils in young children suffering with pneumonia. The entrance of air is to some extent favored by the action of these muscles, but the explanation of their participation in the dyspnœal movements is to be sought in the implication of associated muscle groups in the intense innervation supplied to the essential muscles of respiration.

The term dyspnœa is sometimes used to designate the sensation of breathlessness which attends difficult breathing. Hence *subjective* and *objective dyspnœa*. These two forms are usually associated, but to this statement there are exceptions. Cases occur in which, with persistent obstruction of the respiration and well-marked objective dyspnœa, there is no sense of breathlessness or oppression. Cyanosis may even be present without subjective dyspnœa. As death approaches and objective dyspnœa becomes urgent carbon-dioxide narcosis develops and subjective dyspnœa disappears. There are cases, however, in which objective dyspnœa is slight or absent altogether, yet the patient complains of distressing subjective dyspnœa. To this category must be referred the precordial distress of melancholia and the frequent desire of nervous patients to take a series of deep inspirations. Pure subjective dyspnœa is rare.

Cyanosis attends every obstruction to respiration of high grade, whatever the cause. The blood becomes progressively richer in carbon dioxide and poorer in oxygen. In chronic conditions attended with dyspnœa the organism may gradually become adjusted to subnormal oxygenation of the blood, so that the other functions are fairly well performed and the patient, although manifestly dyspnœic, especially upon exertion, and constantly cyanosed, has little subjective dyspnœa. On the other hand a similar degree of obstruction to respiration, if rapidly established, is attended with the most urgent and distressing dyspnœa.

Pneumothorax affords a striking example of the adjustment of the organism to respiratory disturbances of high grade. The sudden interference with respiration causes intense objective and subjective dyspnœa, which gradually subsides and in many cases wholly disappears so long as the patient is at rest.

Dyspnœa is of much less unfavorable prognosis when the cyanosis is slight than when it is deep and persistent.

Forms of Dyspnœa.—Dyspnœa as the result of pain occurs in pleurisy and especially in diaphragmatic pleurisy, peritonitis, inflammation of the diaphragm, and in affections of the intercostal muscles, as myalgia and trichinosis. Deep breathing is impossible; the respirations are shallow and hurried. The difficulty is not mechanical but functional.

Dyspnœa from Diminution of the Respiratory Surface or Limitation of the Respiratory Excursus.—These conditions are commonly associated. They are present in diseases involving the parenchyma of the lungs, such as croupous and bronchopneumonia, large infarcts, congestion, and the like; also in those affections in which the capacity of the thorax is decreased, as pleural and pericardial effusion, pneumothorax, tumors, massive hypertrophy of the heart, and kyphoscoliosis; and finally whenever the movements of the chest are hindered, as in emphysema, severe

chest-pain, or spasm or palsy of the respiratory muscles. Under these circumstances the tidal air is diminished and the number of respiratory acts is correspondingly increased. In proportion as the requirements of the organism are thus satisfied the associated symptoms,—subjective dyspnœa and cyanosis,—are slight or absent. Bodily effort increases the difficulty. The deficiency of oxygen asserts itself and these symptoms become manifest upon moderate exertion. If the condition be unilateral, as in pleural effusion, vicarious respiration is established.

Dyspnœa in Circulatory Derangements.—Valvular lesions cause dyspnœa when the compensation fails. Myocardial changes act in the same way. There is a transference of blood-pressure from the arterial to the venous side of the circulation. The organs receive less arterial and retain more venous blood than normal. The circulatory derangement affects the respiratory centre, with the result that the breathing is increased both in frequency and depth. Lesions of the left side of the heart cause overfilling not only of the veins of the general circulation but also of those of the pulmonary circuit. The overfilling of the pulmonary capillaries, which ultimately gives rise to brown induration, is an additional cause of dyspnœa, not so much because of the space occupied by the blood as by reason of the impairment of elasticity in the congested tissue of the lung. The alveolar distention remains near the inspiration point and the respiratory excursus is correspondingly diminished. The loss of elasticity acts as a direct hindrance to breathing. The paroxysmal attacks of dyspnœa in such cases constitute so-called cardiac asthma. This term is frequently used to describe any shortness of breath occurring in disease of the heart. It is preferable to restrict it to the attacks which bear a close resemblance to true bronchial asthma. Such attacks often come on at night after the first sleep. In both conditions the form of dyspnœa is the same. There is a tendency to prolongation of the respiratory act with difficult and prolonged expiration attended by stridor. To the habitual overfilling of the pulmonary capillaries in mitral disease must be ascribed the dyspnœa upon exertion which is so common in this condition in the absence of impaired compensation. A further cause of dyspnœa in circulatory disturbances is the bronchial catarrh which is present to some degree in most of the cases.

Dyspnœa in Obstruction of the Upper Air-passages.—The stress upon the inspiratory muscles is proportionate to the degree of obstruction. The respiration tends to become prolonged and deep. In many cases, however, it is increased in frequency and correspondingly superficial. This form of dyspnœa is present in the marked stenosis of the pharynx which occurs as the result of hypertrophy of the tonsils or retropharyngeal abscess, in spasmodic and membranous laryngitis, in œdema or spasm of the glottis, in paresis of the abductor muscles of the larynx (posterior crico-arytenoids) and in narrowing of the pharynx and trachea by tumors, foreign bodies, and compression from outside, as in the case of aneurism or mediastinal tumor.

As the obstruction reaches a high grade the volume of air in the lungs is progressively diminished and the less rigid portions of the thorax yield to the pressure of the external atmosphere. The depressions are especially marked in the epigastrium and the suprasternal and postclavicular regions.

In young children, in consequence of repeated attacks of laryngitis or bronchitis the cartilaginous portions of the wall of the thorax yield and more or less persistent deformities of the chest arise. Among these are the wide, shallow, oblique depressions at the base of the chest known as Harrison's furrows, and the prominence of the sternum, known as chicken-breast.

The stridor in this form of dyspnœa is characteristic. It is commonly loud, prolonged, and hissing or whistling in character and usually much more marked upon inspiration than upon expiration,—a fact that finds explanation in the lateral drawing together of the tissues below the seat of obstruction in consequence of the tendency to vacuum caused by the powerful inspiratory effort and the greater force of the inspiration as compared with the expiration. In fact stridor may be wholly absent during the expiration. When, however, patients suffering from stenosis of the upper air-passages in increasing obstruction become obliged to use the abdominal muscles in active expiration, expiratory stridor becomes marked or predominant.

Dyspnœa in Bronchitis.—Dyspnœa arises, as a rule, only in those cases in which the catarrhal inflammation involves the finer tubes. The lumen is narrowed by swelling of the mucosa and the presence of secretion or exudate. If the narrowing involves a limited number of bronchial tubes the difficulty in breathing is not urgent and compensation takes place by increase in the respiration frequency. When the obstruction involves a great number of bronchial tubes differences in type of the dyspnœa arise which depend upon the degree of obstruction. In so-called capillary bronchitis the respiratory surface is diminished to an extent corresponding with the number and distribution of the lobules involved; dyspnœa with hurried respiration then results. In the dry bronchitis of the middle-sized tubes it is yet possible for a sufficient quantity of air to be drawn into the lungs. This can generally be accomplished best by respiration of diminished frequency and abnormal depth, just as in stenosis of the larynx.

Dyspnœa in Bronchial Asthma.—The breathing is slow, the expiration usually prolonged and accompanied with stridor—*expiratory dyspnœa*. The râles can be heard at a considerable distance from the patient. The difficulty is not to get the air into the lungs but to get it out. Acute emphysema occurs and the respiratory excursus is greatly diminished; hence the "air hunger."

Dyspnœa in Emphysema.—The chest tends to assume permanently the inspiratory form. The elasticity of the lung parenchyma is impaired. The respiratory excursus is correspondingly diminished. The alveolar septa are in many places destroyed, together with the intra-alveolar blood-vessels. The breathing is shallow and frequent. The patient is distressed for breath, especially upon the slightest exertion. The dyspnœa is increased by the bronchitis which is so common in emphysema. Modifications arise in consequence of the frequent occurrence of bronchial asthma in emphysema.

So-called Uræmic Dyspnœa.—This form may occur as a true uræmic bronchial asthma. There is slowing of the respiration with prolonged expiration and expiratory stridor. The condition is not common. The dyspnœa in the majority of the cases is not actually uræmic but rather a manifestation of cardiac derangement, bronchial catarrh, or beginning pulmonary œdema.

The Dyspnœa of Fever.—Rise of temperature is usually associated with increase in respiration frequency. Artificial elevation of temperature also causes hurried breathing. The dyspnœa is doubtless due to the action of heated blood upon the respiratory centre. As it bears no constant relation to the height to which the temperature rises, it is probably due, in part at least, to the action of the fever-producing toxins. Experience has shown that febrile diseases in which the respiration frequency, in the absence of lung complications, is very high, are as a rule of serious import.

The Dyspnœa of Anæmia.—When the hæmoglobin is diminished, the oxygen requirement of the organism demands the most complete performance of the respiratory function. There is no hindrance to respiration and the breathing is quickened and increased in depth. This form of dyspnœa is characteristic of high grades of secondary anæmia such as occur in hemorrhage, in advanced pernicious anæmia, and in chlorosis, following exertion. It is a symptom of internal hemorrhage and occasionally of hemorrhagic pancreatitis.

COUGH AND ALLIED PHENOMENA.

The normal rhythmical expansions and contractions of the thorax serve the physiological purposes of respiration. Certain other movements which are respiratory in character serve other purposes. Of these some are voluntary, others involuntary, some purposeful, others spasmodic. Among such movements are the following:

Cough.—A more or less deep inspiration is followed by an expiratory act which is interrupted by repeated partial closure of the glottis and the production of a series of characteristic sounds. The air is expelled through the narrowed glottis with some force so that foreign bodies, such as a crumb, mucus in the respiratory passages, and the like, are swept from the upper air-passages into the mouth. In the great majority of cases cough, whether in consequence of lesions of the respiratory organs or disease or irritation in distant organs, is reflex and spasmodic. It may be voluntarily produced.

Hawking is a voluntary act, the result of irritation in the pharynx. It resembles cough except that the glottis is open and the expiration continuous.

Sneezing consists in a deep inspiration followed by a forcible expiratory blast through the nose; the glottis is open and the mouth usually but not always closed. Sneezing is excited by irritation of the terminal fibres of the nasal branches of the fifth pair of cranial nerves and is often preceded by peculiar sensations in the nose.

Laughing is an emotional act characterized by deep inspiration succeeded by repeatedly interrupted expiration with an open glottis and vibrating vocal cords. The mouth is wide open, the expiration is much less forcible than in coughing, and the muscles of expression give a characteristic appearance to the face. Laughing may be voluntary or involuntary. When very violent and repeated it may be spasmodic and accompanied by tears.

Crying closely resembles laughing. It cannot in fact always be distinguished from laughing and the one may readily alternate with the other

in young children or patients suffering from hysteria. The rhythm and the facies are different. Crying is involuntary and accompanied by tears.

Sobbing follows long spells of crying or is the expression of deep grief. It is characterized by interrupted inspirations with a partially closed glottis, followed by a prolonged quiet expiration, and is usually involuntary.

Sighing consists in a prolonged inspiration attended by a characteristic soft sound. The mouth is closed or the lips but slightly parted; it is largely voluntary.

Yawning consists in a prolonged deep inspiration through the widely opened mouth accompanied by a peculiar sound. The glottis is open and the expiration short. The arms are thrown out and the shoulders back. It may be either voluntary or involuntary but is not spasmodic.

Snoring occurs during sleep. The mouth is open and the relaxed palate is thrown into vibration by the in- and outflowing air. The sound is louder during inspiration. It is much more liable to occur when the sleeper is on his back.

Stertor or stertorous breathing resembles snoring. It occurs in apoplectic and other comatose states, as cerebral concussion, fracture of the skull, epilepsy, deep anæsthesia, alcoholic stupor, poisoning by opium, illuminating gas, and other narcotics, pulmonary œdema, and all conditions in which excessive amounts of mucus or fluid are accumulated in the bronchi, and frequently in the death-agony. Among the varieties of stertor are *buccal*, characterized by vibrations of the lips and puffing of the relaxed cheeks during expiration; *palatine*, in which the soft palate vibrates with the in-going and out-going air; *pharyngeal*, caused by the sinking back of the base of the relaxed tongue into near relation with the posterior wall of the pharynx; *mucous*, the coarse snoring sound produced by the churning of the respiratory air through fluid, such as mucus or blood in the trachea or larger bronchial tubes.

Stridor or stridulous breathing is that noisy form of breathing caused by obstruction in the larynx or trachea. This symptom may be present in croup and diphtheria, œdema of the glottis, laryngeal tumors, mediastinal new growths, and aortic aneurism. Stridor due to laryngeal obstruction is commonly accompanied by aphonia. It varies greatly in character, being harsh, musical, or crowing.

Hiccough is caused by a sudden spasmodic contraction of the diaphragm accompanied by closure of the glottis. There is a peculiar abrupt sound and a distressing sensation of jerking in the epigastrium. It is due to irritation of the terminal filaments of the phrenic nerve, which may be direct or reflex. It may occur as the result of gastric or peritoneal irritation or may be the manifestation of a derangement of the nervous system. Hiccough is occasionally observed after excessive or injudicious eating or drinking, in gastric disorders, peritonitis, the so-called typhoid state, and uræmia. It occurs also in hysteria and may constitute a pure neurosis. The hiccough of cerebral disease, as hydrocephalus or meningitis, is doubtless the result of irritation of the central origin of the phrenic nerve. The writer knows a gentleman in whom certain kinds of tobacco invariably produce distressing hiccough, while others can be smoked with impunity.

Persistent and intractable hiccough occasionally attends the closing days of fatal illness and in rare instances, occurring as a neurosis, has caused death by exhaustion.

Of all the special or modified respiratory movements cough has the most important bearing upon diagnosis and therefore requires more extended consideration.

SIGNIFICANCE OF COUGH IN DIAGNOSIS.

Etiological Considerations.—Reflex Cough.—Cough in the vast majority of instances is the result of reflex irritation of the terminal nerve filaments of the vagus distributed to the respiratory tract. Irritation of the mucous membrane of the larynx above the vocal cords does not produce cough but causes gagging, while irritation below the cords gives rise to cough. Especially sensitive areas are the interarytenoid space and the region of the bifurcation, while the general mucous membrane of the trachea and bronchi shows a scarcely inferior irritability. Lesions of the lung parenchyma probably do not cause cough, though they are usually connected with pathological conditions of the bronchi. Pleural irritation is commonly attended by this symptom. The aspiration of a pleural exudate is frequently followed by prolonged and violent cough.

Irritation of the nasal mucosa, which is supplied with sensory nerve-twigs from the trigeminus, may in neurotic individuals produce coughing with lachrymation, as in rose cold, hay fever, and similar conditions. In such persons the slightest touch of the probe in the sensitive areas may provoke violent attacks of coughing. Less common is cough as a symptom of hypertrophic, atrophic, or vasomotor rhinitis or of polypi or deviations of the septum. The inhalation of dust or smoke, irritating chemical fumes, as those of ammonia, bromine, or pungent substances, as pepper, produces cough in a normal respiratory mucous membrane. Violent paroxysmal cough is excited by the insufflation of a foreign body, as a crumb or a drop of liquid, into the larynx or through the glottis. The common source of irritation is to be found in a morbid condition of the mucous membrane of the larynx, trachea, or bronchi. There may be merely inflammation and hyperæsthesia with altered or deficient secretion; an exudate of varying consistence, from the thin fluid of bronchorrhœa to the tough masses of tenacious mucus in the early stages of acute bronchitis; or the solid bronchial casts of the terminal tubules in croupous pneumonia or fibrinous bronchitis; or, finally, the material in the bronchi may be derived from adjacent structures and consist of blood, as in bronchopulmonary hemorrhage or an aneurism, or pus from an empyema, a subphrenic abscess, or an abscess of the liver.

Cough Occurs as a Symptom in All Forms of Respiratory Catarrh.—In acute or subacute rhinitis it is often associated with sneezing; in laryngitis with hoarseness or aphonia; in tracheitis or tracheobronchitis with substernal pain; in bronchitis of the larger tubes with tickling sensations in the early stages and a mucopurulent expectoration later; in bronchitis of the smaller tubes with dyspnœa and a tendency to cyanosis; in pneumonia with fever and other indications of acute illness; in pleurisy with a

stitch in the side. Cough is a prominent symptom in bronchiectasis and in all diseases, both acute and chronic, in which the respiratory mucous membrane is primarily in a morbid condition or is irritated by the presence of exudates or discharges from the alveolar tissues or other sources. The irritation is always mechanical, often also chemical. Cough is therefore a constant and suggestive symptom in pulmonary tuberculosis in all its forms and at all stages of its progress. Cough is at once the reflex response to the irritation and the effort to remove the cause of the irritation, and ceases when the effort is successful. The offending substance ejected is known as expectoration, phlegm, or sputum, or, in the plural, sputa.

Exceptionally there are cases in which, with the most pronounced symptoms and signs of disease of the lungs, cough is wholly absent. This may occur in the low fevers, the pneumonia of drunkards, the cachectic, or the aged, in cerebral disease, and shortly before death. The reflexes are obtunded and bronchial secretion or an exudate, the presence of which is manifested by râles, fails to excite cough. The sudden cessation of cough in grave cases of pneumonia or advanced phthisis is an ominous sign. Cough is sometimes absent because the bronchial secretion is swept onward by the ciliated epithelium to the larynx and removed by hawking. If it is then swallowed, as is a frequent occurrence, not only is cough absent but also expectoration.

Much less common is cough due to extrarespiratory irritation. The sufferers are usually neuropathic.

Pharyngeal Cough.—Tickling of the wall of the pharynx or the base of the tongue, which in most persons is resented by gagging, in some is followed by cough. Lymphoid growths in the nasopharynx and collections of thick mucus, or the presence of inflammatory exudates, may be the cause of cough. Elongation of the uvula and paresis of the palate may excite cough by producing irritation of the posterior wall of the pharynx, especially during recumbency.

Ear Cough.—Not infrequently paroxysmal cough is produced by the presence of a foreign body in the external auditory meatus or by disease of that passage. The mere introduction of the speculum may cause cough so violent as to make the examination most difficult. The afferent nerve is the auricular branch of the pneumogastric or, according to others, the auriculotemporal branch of the fifth nerve.

Stomach Cough.—The popular explanation of certain forms of cough as a manifestation of disorders of the stomach is sustained neither by pathological nor experimental investigation. The morning cough of the drunkard is to be accounted for by the pharyngeal catarrh which accompanies chronic alcoholic gastritis; of the consumptive, by lesions of the lungs or larynx, with which secondary gastric disorders are commonly associated. The cough occasionally observed in subacute catarrhal gastritis and which disappears as the gastritis improves is due to the associated pharyngitis. Bronchitis is very common in chronic alcoholism and other forms of ill health with derangement of the gastro-intestinal tract, and a careful investigation of the cases of so-called stomach cough will almost always demonstrate, with the gastric condition, associated lesions of the respiratory tract which account for this symptom.

Liver Cough.—This symptom is doubtless due to irritation of the diaphragmatic pleura. It is met with in certain cases of hypertrophy of the liver, perihepatitis, hydatids, and hepatic and subphrenic abscess.

Disease or enlargement of the spleen may also in rare cases be the cause of cough.

Cough may exceptionally be provoked by pressure in the region of the liver or spleen.

For some years the writer had under observation a case of ventral hernia midway between the tip of the ensiform cartilage and the umbilicus in the median line, in which violent paroxysmal cough attended the presence of the tumor and immediately subsided upon its reduction.

Dentition.—Cough is not uncommon during the first dentition, without manifestations of disease of the respiratory tract. It appears before the eruption of the successive groups of teeth and disappears with the completion of the process.

Mediastinal Cough.—Mediastinal tumor or abscess, thoracic aneurism, enlarged bronchial glands, and caries of the dorsal vertebræ are occasional causes of persistent and troublesome cough. Massive hypertrophy or great dilatation of the heart is also in some instances accompanied by cough.

Nervous Cough.—The diagnosis of nervous cough is only to be made when, in default of direct signs or symptoms or by exclusion, the absence of disease of the respiratory organs or other lesions recognized as the cause of this symptom can be established. Not rarely cough is the only direct manifestation of a bronchitis or pulmonary tuberculous process in which, for the time being, the ordinary physical signs are lacking. It often happens that the diagnosis of nervous cough is made when the intensity and persistence of the cough is altogether out of proportion to its actual physical cause, as is common in neurotic individuals. Nevertheless in some cases cough must be recognized as a purely nervous phenomenon. There are persons who cough whenever their feet are chilled or a cold air blows upon an exposed part of the body. Paroxysmal cough of purely nervous nature is not uncommon in both sexes at puberty. Cough is one of the multitudinous symptoms of hysteria. Under certain conditions cough may occur in neurotic individuals in consequence of disease or irritation of the mammæ or of the genital organs in either sex. It has been shown that cough may be excited by irritation of the floor of the fourth ventricle above the centre for respiration. Whether, under pathological conditions, a true "centric cough" occurs is open to question. Irritation of a "cough centre" has been invoked to explain hysterical and other coughs of purely nervous origin.

Clinical Varieties of Cough.—The *character* of the cough is of importance in diagnosis. It is modified according to the seat of the irritation, whether respiratory or extrarespiratory; by the anatomical structure involved, as the larynx, bronchi, pleura; by the amount and consistence of the irritating substance; and by the constitutional peculiarities of the patient. The following forms demand especial consideration:

Dry Cough.—Patients themselves recognize the distinction between dry and moist cough. Cough due to irritation of the respiratory mucous membrane is dry when it occurs in the absence of secretion or if the secre-

tion is tough, tenacious, and not readily dislodged. Extrarespiratory cough—so-called "reflex cough"—is dry. The sound is hacking, barking, or ringing and is not accompanied by expectoration. Dry cough is frequently spoken of as "unproductive." It occurs in the early stage of acute bronchitis, bronchial asthma, influenza, pneumonia, and pleurisy, in affections of the upper air-passages, phthisis, and pertussis. This is the cough which is excited by the inhalation of foreign bodies, irritating fumes, or dust, and by extrarespiratory causes. It results from pleural irritation and is encountered in pleurisy with fibrinous exudate and upon the withdrawal of an effusion.

Loose or Moist Cough.—This cough is associated with sounds indicating the part played by fluid in the mechanism of its production. It differs from dry cough not only in its acoustic characters but also in the occurrence of expectoration. It is "productive." Loose cough occurs in the later stages of acute bronchitis, influenza, and pneumonia; toward the close of the paroxysms of whooping-cough and asthma; in chronic bronchitis, bronchiectasis, and pulmonary gangrene; in advanced phthisis; and in all conditions attended by moderate or abundant bronchial secretion.

Constant and Recurrent Cough.—Adjectives such as constant, persistent, recurrent, designate peculiarities of the cough dependent upon the persistence or recurrence of its cause. So-called nervous cough is usually persistent; also the cough which attends diseases of the upper air-passages and acute bronchitis and that of bronchorrhœa. On the other hand, in chronic bronchitis, especially when there is bronchial dilatation, the cough is apt to occur paroxysmally at varying intervals. The expectoration of a large amount of matter is followed by relief. After a time the secretion reaccumulates, a mere overflow into the bronchi on change of posture excites cough, and the process is repeated. This form of cough attends the later stages of phthisis with large vomicæ and occurs in some cases of empyema with bronchopulmonary fistula. Recurrent cough is very common in chronic bronchitis and phthisis; it constitutes the "morning cough" of these conditions. The secretion accumulates slowly during sleep without exciting irritation. On waking, the patient moves, the accumulated material shifts its position a little, the bronchial reflex is brought into play, cough results and continues until the offending mass is expelled.

Paroxysmal Cough.—Recurrent cough is not necessarily paroxysmal, and paroxysmal cough for the time may be constant. The word paroxysm conveys the idea of suddenness and intensity. Such is the cough of acute inflammatory conditions; that caused by foreign bodies in the air-passages, the insufflation of saliva, and the like; by the periodical flooding of the bronchi with the abundant contents of the cavities in phthisis, bronchial dilatation, pulmonary abscess, or gangrene; and finally that of pertussis. The paroxysm recurs at intervals varying from an hour or less to once a day or longer. In the case of cavities or a bronchial fistula in empyema the interval is determined by the accumulation; in pertussis by the intensity of the neurosis. In the latter condition the cough is characteristic. Into its production two factors enter: an abundant tenacious mucus and a pathological nervous excitability. After a long inspiration, the expira-

tory cough-efforts succeed each other with such rapidity that inspiration is partial or absent until at last a prolonged inspiration takes place which, by reason of the spasmodic contraction of the glottis, is attended by a harsh, crowing sound or whoop; hence the common term whooping-cough. A somewhat similar inspiratory whoop sometimes attends the paroxysmal cough of other diseases, but so infrequently that in the vast majority of cases the phenomenon justifies the diagnosis of whooping-cough. The differential diagnosis involves consideration of the age of the patient, the presence or absence of an epidemic, history of exposure, the duration and course of the attack, and the presence or absence of lesions capable of causing violent paroxysmal cough other than that of pertussis. The cough which attends enlargement of the bronchial glands, mediastinal tumor, caries of the dorsal vertebræ, enlargement of the heart, and pericardial effusion is paroxysmal and dry. Quite often it has the laryngeal character. Very violent paroxysms of cough frequently result in retching and vomiting and, as a result of the venous congestion from intrathoracic pressure, in hemorrhage from mucous surfaces or into the skin.

Croupy Cough.—A dry cough, described as metallic, ringing, or croupy, is characteristic of laryngeal irritation. The voice is usually hoarse or aphonic, though it may be unimpaired. The laryngeal cough occurs in simple or exudative laryngitis, spasm of the larynx, from the inhalation of smoke or dust, as the result of the irritation produced by foreign bodies in the larynx, and in tuberculous, syphilitic, or cancerous ulceration. The cough of hysteria is usually laryngeal in character, though upon laryngoscopical examination neither swelling nor paralysis may be present. It is described by such adjectives as barking or croaking and resembles other hysterical manifestations by the readiness with which it may be voluntarily produced. A barking laryngeal cough, in the absence of swelling of the laryngeal mucosa or paralysis of the vocal cords or of lesions directly or indirectly involving the recurrent laryngeal nerves, is commonly hysterical.

Suppressed Cough. — Voluntary efforts to suppress cough are made under circumstances in which the sound of the cough is likely to annoy others and when the act is attended by pain, as in pleurisy, acute peritonitis, and some forms of acute bronchitis. The suppressed cough is usually lacking in tone, and is explosive and persistent.

Undeveloped Cough.—Incomplete efforts at cough, unattended by the characteristic sound, are observed in cases of destructive ulceration or paralysis of the vocal cords or of partial paralysis of the expiratory muscles. This form of cough is encountered in laryngeal phthisis, in patients suffering from bulbar paralysis, in enormous ascites or abdominal tumors, and in conditions attended with extreme debility, especially the later stages of croupous and bronchopneumonia, chronic bronchitis, pulmonary œdema, and consumption.

In the majority of instances the diagnostic significance of the symptom cough is direct and obvious. It is the indication of disease affecting the respiratory organs, manifested more or less fully by concurrent signs and symptoms; in a far smaller proportion of cases its significance is remote and obscure and only to be learned by close and systematic study

of the various organs or parts to derangements of which it may be due. Important among these derangements are diseases of the intrathoracic circulatory organs, mediastinum, ears, teeth, and nose, some nervous affections, and the neurotic constitution. To this list must be added malingering, since cough may be a voluntary act.

THE EXPECTORATION OR SPUTUM.

These terms are applied to material voided by coughing or hacking. The expectorated substance is usually a secretion or exudate derived from the mucous membrane of the nose, pharynx, larynx, or bronchial tubes, or from the alveoli. It may consist of pus, which finds its way into the air-passages from an abscess or an empyema, or of blood from the pulmonary vessels or an aneurism. With these substances are frequently admixtures of food, drink, and the secretions of the mouth. Macroscopic and microscopic foreign bodies which have found their way into the respiratory passages are usually voided in the sputa.

Any of these substances may be present and not expectorated. Infants and young children almost always swallow the sputa and older persons frequently do so as a habit or from inability to expectorate or in abnormal mental states.

The naked-eye examination of the expectorated matter is frequently of great use in diagnosis; the microscopic examination is often essential. For the ordinary bed-side examination a considerable quantity of the sputum should be collected, preferably in a transparent glass spit-cup.

The quantity of the sputum varies according to the nature of the pathological process. Persistent and distressing cough may yield only an occasional small tough mass of tenacious material, as in dry bronchitis or beginning phthisis. In other patients an occasional spell of coughing may bring up enormous quantities of material, as in some forms of chronic bronchitis, bronchiectasis, advanced phthisis, pulmonary œdema, and hæmoptysis. The amount of pus expectorated in empyema with bronchopulmonary fistula may exceed 1000 c.c. in twenty-four hours.

The consistence bears some relation to the amount. An abundant expectoration is usually more fluid than a scanty one. Sputum composed of blood, pus, or a serous fluid is always thin; that consisting of mucus or mucopus usually thick and frequently tough and tenacious.

The reaction of fresh sputum is commonly alkaline. After standing for some hours in the cup, the sputum yields an acid reaction—a change due to decomposition processes caused by bacteria.

The color and translucency vary with the nature of the disease. Mucous expectoration may be transparent and thin, resembling saliva in consistency, or much thicker and still transparent. In proportion as cellular elements are present the sputum becomes thick and opaque, assuming the yellowish or greenish-yellow hue of pus. The gradations are expressed by the terms mucous, mucoid, mucopurulent, and purulent expectoration. Serous expectoration is usually clear and transparent, sometimes slightly tinged with blood. It is thin, frothy, and abundant, and occurs in œdema of the lungs and in the rare cases of perforation of a

serous pleural exudate. The albuminous expectoration which exceptionally follows the aspiration of a pleural exudate is also thin, colorless, and abundant. The color is red when the sputum is admixed with blood. The proportion varies from pure blood to a mere trace sufficient to impart a faint pink tinge. Hemorrhagic sputum occurs in traumatism of the lungs, in the blood-spitting of tuberculosis, in pulmonary infarct, and in croupous pneumonia. It is also present in cases of gangrene of the lung, tumor of the lung, and intense pulmonary congestion. The "rusty sputum" of pneumonia owes its varying shades of color to derivatives of the blood-coloring matter. In rare instances the sputum of pneumonia is lemon-yellow or grass-green. These variations suggest the changes in color that take place in the subcutaneous blood extravasations following a bruise. In the adynamic and septic forms of croupous pneumonia and, more rarely, in gangrene of the lungs the expectoration is fluid and dark colored. This form of sputum is described as "prune-juice" expectoration. The sputum in malignant disease of the lungs is often viscid, tenacious, and of a bright red color. This is the "currant-jelly" sputum of authors. A still more objectionable term is "anchovy-sauce" sputum—a term applied to brownish-red sputum such as is seen in rupture of a liver abscess through the lungs, the peculiar appearance of which is due to the mixture of altered blood, pus, and bile.

Blood-streaked sputum may occur in the following conditions: violent cough, acute bronchitis, or disease of the mitral valves. It may result from the admixture of blood from the mouth, as in the case of scurvy and other forms of inflammation of the gums with bleeding, or of ulceration of the tonsils or pharynx, or from the oozing of blood from an aortic aneurism into a bronchus. It occurs also in acute bronchopneumonia and plastic bronchitis. It is very often observed a day or two after an attack of hæmoptysis. Under these circumstances the streaks or masses of blood are clotted and dark. Blood-streaked sputum is not uncommon during the course of pulmonary phthisis.

Yellow or green sputa can only be regarded as deriving their color from altered bile pigment when icterus or at least yellowness of the conjunctiva and biliary pigment in the urine are actually present. Icteric sputum may occur not only in pneumonia complicated with jaundice but also in any form of lung disease in a patient suffering from jaundice. A peculiar brownish tint is sometimes seen in the sputum in cases of chronic valvular disease. It is due to the presence of amorphous pigment in the epithelial cells. The brownish sputum sometimes seen in pulmonary abscess and other destructive processes involving the lung owes its color to the presence of hæmatoidin crystals, which are also the source of the coloring matter in the ochre-yellow purulent sputum of liver abscess with perforation into the lung. Greenish sputum is sometimes encountered in sarcoma of the lungs and very rarely in carcinoma. Remarkable coloration follows the habitual inhalation of certain dust-particles. Black sputum is common in those who breathe an atmosphere laden with coal-dust or soot. The pigment particles are only to a limited extent free in the sputum; much more commonly they are enclosed in round or oval cells which are in part epithelial, in part leucocytes.

The color of the sputum varies in different forms of pneumonoconiosis. In anthracosis it is often of an intense black; in the siderosis of mirror polishers it may be ochre-red; workers in lapis lazuli may have a blue sputum, and so on. The dust particles which are expectorated are those recently inhaled which have not yet penetrated to the lung parenchyma, as is shown by the fact that the color disappears from the sputum in the course of a short time after the workman has abandoned his occupation. If however the color persists or returns after a time, it is the sign of a destructive process, usually tuberculous. Various colors may be imparted to the sputum by articles of food or drink, as milk, wine, coffee, or medicines. Finally after the sputum has been ejected it may undergo color changes in consequence of the growth of chromatogenous bacteria and thus become blue, green, yellow, or red. The Bacillus pyocyaneus may be the cause of a blue discoloration of the sputum.

Air.—Air in the sputum is shown by the presence of minute bubbles. The quantity depends upon circumstances. It is greater in sputum from the finer than in that from the larger tubes, in sputum of thin than in that of thick and tenacious consistency, and in the sputum which is largely composed of mucus than in that which is chiefly pus. A little water in the spit-cup enables us to estimate the relative amount of air, as it affects the specific gravity; sputa which float contain air; those which sink do not. The sputa of phthisis and bronchitis often present the appearance of flat circular or coin-shaped masses—the so-called "nummular sputa"—or the masses may be globular; they are commonly grayish-white and sink in water; sometimes they are buoyed up by the small bubbles of air which they contain.

Stratification.—Layer formation takes place in the collected sputa of chronic bronchitis with abundant expectoration—bronchorrhœa—of bronchiectasis, putrid bronchitis, and gangrene of the lungs. The material is of thin consistence and abundant. As a rule it collects in three well-defined layers which can be studied by the use of a glass spit-cup. The upper stratum contains air and is often frothy, the middle is fluid and consists of mucus or pus-serum, and the lower is sedimentary and made up of pus corpuscles, molecular lung detritus, and shreds of necrotic tissue.

Odor.—The odor of fresh sputum has, under ordinary circumstances, nothing characteristic. Speedy decomposition renders it offensive. The sputum of putrid bronchitis, bronchiectasis, gangrene of the lung, and perforating empyema is always heavy and fetid; frequently horribly offensive. In abscess of the lung and in many cases of advanced phthisis also it is offensive. The foulness is imparted to the expired air, which not infrequently is even more obnoxious than the sputum. It is probable that in the cases of pulmonary consumption in which the sputum and breath are foul there is already cavity formation, though too small in some instances to be recognized by the methods of physical diagnosis, in which the secretion collects and undergoes decomposition. Very often the odor is imparted to the breath by offensive material in the crypts of the tonsils or by decaying teeth or other necrotic material in the mouth—a fact that cannot in all cases be established by the use of deodorizing mouth washes, since they act only upon the surfaces with which they come in contact and cannot reach deeply-seated tissues from which the odor may proceed.

Other Macroscopical Characters of the Sputum. — Very often the expectorated material presents a homogeneous appearance, as is the case with mucus, pus, blood, etc. Occasionally, on the other hand, the matter expectorated at different times varies in appearance and not infrequently a single mass consists partly of mucus and partly of pus, or of these substances with masses of blood. The purulent expectoration of an empyema or a pulmonary abscess is sometimes flaky or thready, best shown when the sputum is suspended in water. The naked-eye characters of the sputum may be conveniently studied by pouring a small quantity upon a plate or slab of which half is black, the other half white, or placing a specimen between two glass plates and examining it over a white and black background alternately. A hand lens may be used and particular objects removed for microscopical examination. Minute, dirty gray masses of necrotic lung tissue containing elastic fibres may be detected in the specimen in gangrene and abscess of the lung and in the later stages of phthisis; fragments of necrotic cartilage in destructive processes involving the bronchi, the trachea, or the larynx, and in rare cases shreds of tissue from tumors of the bronchi or lungs. Minute, dirty white or yellowish masses, in some instances constituting casts of the smaller bronchial tubes, are seen in fetid bronchitis and gangrene of the lungs. These masses consist of aggregations of bacteria and crystals of the fatty acids. They have an intensely disagreeable odor. Similar masses may be expectorated in lacunar tonsillitis and are sometimes present in the crypts of the tonsils in the absence of inflammation. Curschmann's spirals are visible to the naked eye and may be studied with the lens. They consist of twisted masses which may reach 1 or even 2 cm. in length and have a diameter of about 1 mm. These masses are made up of a highly refractive central undulating core or thread around which are coiled spiral filaments which are sometimes branching. The central core was at one time thought to be fibrinous, but has more recently been shown to consist of a substance analogous to mucin. These spirals are formed in the finest bronchial tubes as the product of an exudative bronchiolitis, and as this pathological process is frequently associated with bronchial asthma the spirals are very often found in that disease and in well-marked cases are sometimes present in great numbers. The association, however, is by no means constant; cases of asthma are occasionally encountered in which no spirals can be found in the sputum, and the spirals are sometimes present in the expectoration of cases of bronchitis unattended by asthmatic symptoms. Curschmann's spirals occasionally appear also in the sputum of croupous pneumonia and are then seen to be in strong contrast with the fibrinous casts of the bronchioles which occur in that disease. They have also been encountered in the sputum of pulmonary phthisis. Microscopically, leucocytes, notably eosinophiles, epithelial cells, and Charcot-Leyden crystals are found entangled in the spirals.

Fibrinous coagula, recognizable by their white or grayish-white color, tough consistence, and characteristic form, are found in the sputum under varying pathological conditions. They are usually coughed up in masses surrounded with mucus and, when of great size, with difficulty. In diphtheria fibrinous pseudomembrane is expectorated, sometimes in irregu-

lar masses, sometimes as a fibrinous mould, more or less incomplete, of the larynx or trachea. When the diphtheritic exudate extends to the bronchi, branching casts are sometimes coughed up. These casts may be easily recognized in the sputum and are of great importance both in diagnosis and prognosis. Fibrinous casts are common in croupous pneumonia, in the sputum of which they are frequently present in great numbers. They can be readily seen when the sputum is shaken with water in a test-tube, or when the masses of mucus in which they are embedded are shaken out in water with a forceps. In pneumonic sputum the fibrinous casts are small. Similar casts consisting chiefly of mucus are characteristic of so-called fibrinous or croupous bronchitis and provoke the intense paroxysmal cough of that disease.

Foreign bodies that have found their way into the air-passages by aspiration are usually expectorated promptly. They may, however, remain in a bronchus for a long time and give rise to symptoms of varying intensity. Instances are recorded in which a tooth, cherry-pits and other seeds, a beard of wheat, etc., have been expectorated after periods of months or years. Bronchial concretions, consisting in the main of lime salts and sometimes of considerable size, are in rare instances found in the sputum. They occur only in chronic conditions and are formed in the cavities of phthisis and bronchiectasis, or consist of fragments of bronchial glands that have undergone calcareous degeneration and found their way into the bronchial system. Even more rare is the presence in the sputum of echinococcus daughter cysts, membranes, or hooklets, which have found their way from the lung, pleura, or the liver into the bronchi.

The Sputum in Different Diseases.

Bronchitis.—The sputum is usually mucoid and mucopurulent. As a rule, at the beginning of an acute bronchial catarrh the bronchial secretion is diminished and the sputum scanty. In the course of some days, as the symptoms ameliorate, the expectoration becomes more abundant, less tenacious, and distinctly purulent. As the general symptoms improve there is a gradual diminution in the quantity of the sputum. In chronic bronchitis the expectoration varies greatly; sometimes it is more, sometimes less purulent. The subjective sensations of the patient are usually better when the sputum is of moderate amount, worse when the expectoration is suppressed or greatly increased in quantity (see bronchitis).

Fibrinous or Croupous Bronchitis.—The sputum differs from that of ordinary bronchitis in that from time to time it contains fibrinous casts associated with blood. Charcot-Leyden crystals are also present. The expectoration of the larger casts very often takes place after distressing cough, recurring in paroxysms which are separated by periods of urgent dyspnœa.

Pulmonary Tuberculosis.—The sputum of tuberculosis presents to the naked eye nothing characteristic. All varieties of sputum that occur in ordinary bronchitis, from mucous to purulent, occur in phthisis. In advanced ulcerative phthisis purulent expectoration is often constant and abundant. For the provisional diagnosis the presence of the minute

grayish masses which frequently contain colonies of tubercle bacilli is important. Very often the sputum has an offensive odor; this is especially the case when there are cavities, the contents of which undergo stagnation and decomposition. A positive diagnosis rests upon the presence of tubercle bacilli and, in the absence of other destructive pulmonary lesions, the presence of elastic fibres. It is important for the student to bear in mind that there is no constant relation between the abundance of these morphological elements and the intensity of the process, therefore the gravity of the prognosis. There are cases of pulmonary tuberculosis of the gravest character in which neither tubercle bacilli nor elastic fibres are found. Very often these are cases of *phthisis florida* or of *disseminated miliary tuberculosis* in which the constitutional symptoms develop in advance of the local manifestations. The abundant catarrhal secretion, so common in unfavorable cases, proportionately diminishes the number of tubercle bacilli present in single specimens. On the other hand tubercle bacilli and elastic fibres are frequently found in the early stages at a period when the physical examination of the lung yields vague and uncertain signs. The diminution or temporary disappearance of tubercle bacilli and elastic fibres from the sputum cannot be regarded as indicating a favorable progress of the case in the absence of the general clinical indications of an arrest of the process, such as diminished cough, improved appetite, gain in weight, and disappearance of fever. In a suspected case the presence of tubercle bacilli in the sputum justifies a positive diagnosis. Their absence cannot be regarded as conclusive until repeated examinations have been made.

Acute Miliary Tuberculosis.—The sputum is that of ordinary catarrhal bronchitis and does not contain tubercle bacilli except when there is an associated ulcerative phthisis. In a large proportion of the cases there is no expectoration.

Croupous Pneumonia.—Hemorrhagic sputum is characteristic. Blood-spitting may be the initial symptom. At first the sputum is commonly mucoid, transparent and homogeneous; after twenty-four hours it is blood-tinged and viscid so that it adheres to the bottom of the spit-cup when turned upside down, and sometimes has to be wiped from the lips or face of the patient. At first red from unchanged blood-coloring matter it gradually becomes rusty or orange-yellow in color. Occasionally the sputa are variable; sometimes mucoid, sometimes blood-streaked, at other times pure blood. When jaundice is present the sputum may be green or yellow from the presence of bile pigment. Very commonly the sputum contains fibrinous casts of the smaller tubes. If there is an associated bronchitis of the smaller tubes the typical pneumonic sputum may be modified by the presence of mucus or mucopus. Fluid sputum of a dark brown color—the so-called "prune-juice" expectoration—is an unfavorable sign since it may indicate a beginning œdema of the lungs. In some instances a diminished consistency of the sputum marks the beginning of resolution. The amount of sputum in croupous pneumonia is very variable. In children and the aged, and in adynamic cases, there may be none, and exceptionally it may be scanty in classical cases in adults. A quantity amounting to 200–500 c.c. in twenty-four hours is not uncommon. The amount after the crisis, abundant at first, gradually diminishes.

In some cases there is at this period little or no expectoration. Under the microscope are seen leucocytes, erythrocytes, mucous corpuscles, epithelial cells, and occasionally hæmatoidin crystals. The pneumococcus of Weichselbaum and Fränkel is present in the vast majority of cases, and sometimes Friedlander's bacillus. Fibrinous casts of the bronchioles and moulds of the alveoli are not uncommon. Chemically the expectoration is particularly rich in sodium chloride.

Bronchopneumonia, Including Aspiration Pneumonia and Hypostatic Pneumonia.—The sputum usually presents the appearance of the ordinary forms of bronchitis; exceptionally that of croupous pneumonia. The latter is intelligible, since not only in the clinical phenomena but also in the histological findings there are cases of bronchopneumonia which are difficult to distinguish from croupous pneumonia. In these cases the sputum is hemorrhagic and contains fibrinous exudate. Bacteriologically a mixed infection is the rule. The pneumococcus and Friedlander's bacillus are found in association with the ordinary pus-producing and other organisms. The Klebs-Löffler bacillus is present when the lesions are secondary to diphtheria. In the lobular forms the streptococcus is the common organism; in the lobar forms, the pneumococcus.

Gangrene of the Lungs.—The intensely offensive odor, abundance, fluidity, and dark, dirty, greenish-brown color are characteristic. Upon standing the sputum separates into three strata—an upper frothy layer, which may contain necrotic particles of lung tissue which float by reason of entangled air, a middle thin layer, and a greenish-brown sediment which consists in part of leucocytes, in part of gangrenous detritus. Shreddy fragments of lung tissue of considerable size and frequently showing the alveolar arrangement may be picked out if the sediment is spread upon a glass. Under the microscope are seen elastic fibres, pigment granules, crystals of the fatty acids, cholesterin, leucine and tyrosine crystals, bacteria, and leptothrix. Altered blood-corpuscles are also present. When the fluid is retained in the gangrenous cavity for some time, the elastic fibres may undergo solution owing to the action of a peptonizing ferment. The odor is the more intense in proportion as the communication between the gangrenous areas and the bronchi is more free. Cases occur in which, in the absence of odor during life, circumscribed areas of gangrenous lung have been found upon post-mortem examination.

Abscess of the Lung.—The sputum is essentially purulent. It is offensive, but less intensely so than that of gangrene. When placed in water it has a thready or granular appearance. When the perforation is small there is an accompanying catarrhal bronchitis and the sputa are mucopurulent. When, however, the abscess discharges abruptly, a large amount of pus commingled with masses of necrotic lung tissue and containing elastic fibres in abundance is discharged. Microscopically the sputum contains hæmatoidin, cholesterin and fat crystals and various bacteria.

Perforating Empyema.—The sputum resembles that of pulmonary abscess. It may be at first free from odor but in the course of a little time becomes offensive. It is voided in considerable quantities at varying intervals. Elastic fibres are wholly absent or are present in small numbers. Hæmatoidin and other crystals and pyogenic bacteria are present.

Putrid Bronchitis.—The expectoration presents characteristics similar to that of perforating empyema. It is purulent and foul-smelling, but does not contain elastic fibres. It is voided from time to time in moderate amounts; not in large bulk at intervals of some hours as is the case in empyema with bronchopulmonary fistula and bronchiectasis.

Bronchiectasis.—In saccular bronchiectasis the sputum is sometimes mucopurulent, sometimes purulent. It is brought up from time to time in severe paroxysms and in large quantities—mouthfuls. These paroxysms may follow change of posture, the cough reflex being excited by the shifting of accumulated secretion from the dilatation to the normal bronchial tube. A paroxysm usually occurs in the morning. The color of the expectorated matter may be gray or grayish-brown. It is usually fluid, acid-smelling, sometimes extremely fetid. Upon standing it separates into three layers, an upper consisting of brownish froth, a middle thin watery layer, and a lower, thick and granular. Microscopically the sputum consists of pus corpuscles, epithelial cells, erythrocytes, and large numbers of crystals of the fatty acids. Hæmatoidin crystals are sometimes seen. In the absence of bronchial ulceration, elastic fibres are not found, nor are tubercle bacilli present. Nummular sputa are uncommon. In many cases the sputum cannot be distinguished from that of a putrid bronchitis. Hemorrhage occasionally occurs.

Œdema of the Lungs.—The sputum is usually thin, frothy, colorless or slightly blood-tinged, and abundant. Upon standing it deposits a sediment consisting in part of red blood-corpuscles and in part of elements characteristic of the antecedent condition, as bronchitis or pneumonia. It is largely made up of blood-serum and is therefore rich in albumin. In the rare cases in which perforation of the lung occurs in serofibrinous pleurisy the expectorated matter resembles that of pulmonary œdema but is richer in albumin. A very abundant sputum, similar in character, is sometimes expectorated after paracentesis thoracis, beginning toward the close of the operation—the *expectoration albumineuse* of the French. This serous sputum is the result of an acute pulmonary œdema following the dilatation of the compressed lung.

Bronchopulmonary Hemorrhage—Hæmoptysis.—In the blood-spitting which follows traumatism, the rupture of an aneurism, the lesions of tuberculosis, or new growths involving the lungs the sputum consists of more or less abundant, bright red, frothy blood. The distinction between venous and arterial blood cannot be made, since the dark blood of the pulmonary arteries becomes oxygenized and frothy during its course through the bronchial tubes. The differential diagnosis between hæmoptysis and hæmatemesis rests upon the following facts: In bronchopulmonary hemorrhage the blood is coughed up. In gastric and œsophageal hemorrhage it is vomited, but the account of the patient or his friends is not always satisfactory; in the excitement and alarm the distinction may not be made. Moreover violent paroxysmal cough may on the one hand be followed by gagging and vomiting, while on the other hand some portion of vomited blood may be drawn into the larynx by aspiration and thus excite coughing. The examination of the blood itself is important. Bright red, frothy blood may usually be referred to a lesion of the

respiratory tract; blood that is dark, clotted, and free from air-bubbles, to the digestive tract. But there are exceptions to this rule. In profuse hemorrhage from the stomach the blood is sometimes vomited so rapidly that it is bright red and fluid, while in abundant pulmonary hemorrhage, resulting from erosions of a large branch of the pulmonary artery, the expectorated blood may be dark in color and contain but little air.

The reaction of the blood in hæmoptysis is alkaline. In hæmatemesis which occurs during digestion, when the stomach contains a large amount of acid fluid, the reaction may be acid. Too great importance cannot be ascribed to the reaction of the blood in doubtful cases, since vomited blood is frequently alkaline. The presence of particles of food in the blood is of importance in diagnosis.

There are, however, cases in which the distinction between hæmoptysis and hæmatemesis cannot be immediately made.

The condition of the patient prior and subsequent to the bleeding is in doubtful cases of greater importance than the appearance of the blood. A history of gastric symptoms before the blood loss or the occurrence of such symptoms subsequently is common in bleeding from the stomach. The presence of altered blood in the stools after the hemorrhage points to bleeding from the stomach rather than from the lungs. On the other hand the mere fact that the patient has suffered for some time from cough and expectoration is suggestive of pulmonary hemorrhage, which is apt to be followed for some days by the occasional expectoration of small blood-clots or of sputum mixed with blood. When due consideration is given to these facts errors of diagnosis are not likely to occur.

Hemorrhagic sputum is occasionally encountered in acute bronchitis. This sputum is to be distinguished from pneumonic sputum by the fact that the blood is present in streaks rather than as a homogeneous mixture. Profuse hæmoptysis rarely has its seat of origin in the larynx or trachea, since the blood-vessels of these organs are of relatively small size. On the other hand, blood-streaked sputa are not uncommon in acute catarrhal inflammation of the trachea, larynx, or pharynx. There are forms of hemorrhagic bronchitis characterized by blood-tinged sputum which continue for some days or weeks. Such cases are not uncommon during epidemics of influenza. It sometimes happens, especially during sleep, that the blood in epistaxis trickles into the pharynx and is swallowed. If vomited, such blood may be regarded as due to gastric ulcer. If the blood in the pharynx under these circumstances excites cough and is ejected mingled with mucus, it may be erroneously regarded as coming from the lungs. If the trickling blood be seen upon the wall of the pharynx the diagnosis is at once established and the precise site from which it comes may be determined by means of the rhinoscope.

Infarcts.—The sputum in hemorrhagic infarct is commonly dark in color and resembles pure blood, from which it differs in its somewhat tenacious consistence, suggestive of pneumonic sputum. In point of fact the sputa in cases of pulmonary infarct may vary according to the amount of bronchial secretion present from pure blood to a tenacious blood-tinged mucus.

Chronic Valvular Disease.—Hemorrhagic sputum occurs in certain cases of valvular disease of the heart, particularly in mitral stenosis.

VI.

CIRCULATION; PULSATION; RADIAL PULSE; ANOMALIES OF THE PULSE; CAPILLARY PULSE; VENOUS PULSE.

CIRCULATION.

The term arterial pulse is used to designate the rhythmical fluctuations of the arterial pressure which correspond to the contractions of the ventricles of the heart. These rhythmic fluctuations depend upon the intermittent injection of blood from the ventricle to the aorta, upon the resistance to the arterial flow produced by friction, and upon the elasticity of the walls of the arteries. After the blood enters the capillaries the pressure is no longer intermittent, but is continuous, and pulsation under normal conditions disappears. The pulse may be affected by changes either in the force of the ventricular contractions, in the elasticity of the arteries, or in the peripheral resistance, and by varying combinations of these modifications. The examination of the arterial pulse is therefore obviously of great diagnostic importance. By this means conclusions may be reached in regard to a wide range of clinical facts, including the innervation of the heart, the power of the heart muscle, the blood-pressure, the blood loss in hemorrhage and anæmia due to other causes, the condition of the peripheral arteries, the action of fever-producing toxins upon the heart and blood-vessels, and finally, under certain conditions, in regard to the presence and nature of valvular lesions.

PULSATION.

Arterial pulsation may be studied in any of the superficial arteries. The methods employed in ordinary clinical work are palpation and inspection. Auscultation is of more limited application in the study of the blood-vessels. The results obtained by the use of the sphygmograph are of more value in clinical research and for purposes of record and comparison than for diagnosis.

The increase in the contents of the arterial system which causes the pulsation is accompanied not only by an increase in the tension of the artery at any given point but also by an increase in the length of the vessel. This increase in length results in a more or less marked lateral undulation and exaggeration of the curves of the vessel, normally not sufficient to attract attention, but conspicuous in the temporal arteries of emaciated persons and at various points in the course of the superficial arteries in conditions, such as aortic insufficiency, which are attended with cardiac hypertrophy and relaxation of the arterial walls. The arterial pulse, corresponding to a contraction of the ventricles, is not perceptible at the same moment at all parts of the body, an appreciable interval separating the cardiac impulse, the radial pulse, and that of the dorsal artery of the foot.

The Aorta and Its Branches.—Pulsation in the notch of the sternum is occasionally seen in aged persons in the absence of disease. It occurs in dilatation of the aorta and is a sign of aneurism of the transverse portion of the arch. In rare cases it is due to an anomalous distribution of the branches of the aorta in this region.

Pulsation at the root of the neck is common in cardiac hypertrophy and dilatation, in aortic insufficiency, and in neurotic and anæmic conditions, especially during periods of physical or mental excitement. It is a prominent symptom of exophthalmic goitre. Under these circumstances pulsation of the aorta is associated with a heaving impulse in the innominate and carotids, communicated to the overlying tissues, so that throbbing in this region becomes a sign of importance. It is often accompanied with distention of the veins and flushing of the face.

The differential diagnosis between dynamic dilatation of the arch of the aorta and aneurism cannot in all cases be made during life. Not rarely when the signs of dilatation of the arch and enlargement of the innominate and right carotid have been well marked clinically, the vessels have been found post mortem to be of normal measurement.

Pulsation of the subclavians occurs in the general pulsation at the root of the neck, above spoken of. It is usually less marked than that of the innominate and carotids. Visible pulsation of the subclavians is sometimes present in consolidation and retraction of the lung in phthisis.

Pulsation of the abdominal aorta is very common. It may often be made out in quite thin persons under normal conditions both by inspection and palpation. Under these circumstances it is of very slight intensity. More vigorous pulsation in the line of the abdominal aorta, namely, in the median line or slightly to the left of it, and in the epigastric zone is an important sign of disease. Objectively the pulsation varies in degree. It is frequently violent and throbbing and may be demonstrated by the motion communicated to the stethoscope lightly pressed upon the surface. Subjectively the sensation of throbbing is annoying and frequently distressing. It often prevents sleep. Epigastric pulsation is not in all instances due to the movements of the aorta. It may be directly due to the heart. A faint pulsation in the region of the ensiform cartilage occurs in physiological over-action of the heart, in hypertrophy and dilatation of the right ventricle, and in displacement of the heart towards the right in consequence of left-sided pleural effusion or of emphysema. In the last named condition the epigastric pulsation is often marked, since the heart is displaced toward the median line and the right ventricle is hypertrophied. The pulsation is transmitted to the left lobe of the liver. It is more marked in the neighborhood of the ensiform appendix and costal cartilages than toward the umbilicus, and nice observation will show that it corresponds in time to the cardiac systole, whereas aortic pulsation is slightly postsystolic.

The most common causes of pulsation of the abdominal aorta are referable to the nervous system—simple dynamic pulsation. The throbbing may be a direct manifestation of neurasthenia or hysteria, or it may be a reflex manifestation of disorders of the gastro-intestinal tract. It is

much more common in females and in early life. It occurs also as the result of diminution of the amount of blood and thus becomes one of the signs of anæmia due to hemorrhage or other cause. Marked epigastric pulsation frequently occurs as a sign of enlarged lymphatic glands, carcinoma of the stomach or pancreas, or other form of tumor overlying the aorta. In rare instances fecal accumulations in the colon transmit the aortic impulse to the surface. Thorough evacuation of the bowels is an imperative preliminary measure in the diagnosis of doubtful cases. Finally it may be due to an aneurism.

The diagnostic significance of this sign varies greatly and in some cases is only to be determined by careful study of the associated clinical phenomena. In simple dynamic pulsation the aorta may in thin persons frequently be felt to be somewhat dilated, especially during the paroxysm, but no distinct tumor formation can be recognized. The symptoms of neurasthenia or the stigmata of hysteria are present and these are often associated with gastro-intestinal symptoms. The throbbing is intense and distressing, sometimes diffused but never distinctly expansive. It can be felt when the patient is in the knee-elbow posture. The throbbing of anæmia is much less marked. Pulsation transmitted from the aorta through an overlying tumor communicates a lifting sensation to the hand upon palpation, is usually circumscribed, not expansile, and disappears when the patient is examined in the knee-elbow position, the mass falling away from the aorta under the action of gravity. The clinical phenomena of the primary condition are usually more or less well defined. Errors of diagnosis not infrequently occur under these circumstances, the tumor being mistaken for an aneurism. When well defined the pulsation of an abdominal aneurism is characteristic. If the aneurism be of large size there is dulness continuous with that of the left lobe of the liver. In thin persons a distinct tumor may be felt, the pulsation is expansile and forcible, and persistent rather than paroxysmal. A systolic murmur is very commonly heard in the absence of pressure of the stethoscope or the murmur may be audible in the back. In some cases a low-pitched soft diastolic murmur is heard. In many cases there is a distinct systolic thrill. Both the murmur and thrill may occur in other conditions which cause an abrupt narrowing in the lumen of the aorta, and may be produced by the pressure of the stethoscope. These signs are occasionally encountered in the epigastric pulsation of nervous diseases and in tumors of various kinds developing in relation with the abdominal aorta. The diagnosis of aneurism must therefore be made with extreme caution. It is justified in cases in which there is a distinct tumor with expansile pulsation persisting in the knee-elbow posture and when radiating pain, vomiting, and retardation of the femoral pulse are present. The pulsation of an abdominal aneurism may be manifest in the left hypochondrium or lumbar region. The X-rays furnish an important aid to diagnosis in doubtful cases. Epigastric pulsation must not be confounded with the purely subjective sensation of fluttering in the left hypochondrium of which hysterical women frequently complain. These two phenomena are entirely distinct, though they are frequently present in the same case.

RADIAL PULSE.

The pulse may be studied in any superficial artery. For this purpose the radial, because of its accessibility and convenience, is usually selected. This artery is palpated over the flat portion of the radius between the styloid process and the tendon of the radialis internus. In an anomalous distribution of the artery the radial pulse must be sought for elsewhere. It is a good plan to compare the pulse in the radials of both sides. It occasionally occurs that a small arterial twig occupies the usual position of the radial while the main branch has an anomalous course. In the absence of comparison with the other side an erroneous conclusion as to the volume and force of the pulse would be formed. In any case of doubt the pulse in the bend of the elbows or in the brachial or axillary arteries upon the two sides may be compared. Pathological differences in volume, force, and time, that is to say, retardation upon one side, are due to the interference with the flow of blood in the artery caused by endarteritis and aneurism, or the pressure of a tumor upon the wall of the vessel. Complete obliteration results from embolism or thrombosis. In traumatism from extensive crushing or laceration it is a sign of destruction of the artery. Retardation of the femoral pulse upon both sides may occur in aneurism of the thoracic or abdominal aorta. On one side it is commonly the sign of aneurism of the common iliac artery. Under certain circumstances it is convenient to study the pulse in the temporals, carotids, or even in the posterior tibials.

The best method of feeling the pulse consists in the application of the tips of three adjacent fingers, that of the index finger being, according to an old rule, nearest the heart of the patient. Under changing pressure the distention of the artery which constitutes the pulse is recognized and studied. The value of the pulse in diagnosis depends largely upon the experience and judgment of the physician. In the study of the pulse the following points require especial consideration; (a) condition of the arterial wall; (b) frequency; (c) rhythm; (d) volume; (e) celerity; (f) tension; (g) dicrotism.

The condition of the arterial wall enables us to form conclusions as to the presence or absence of general arteriosclerosis, and to recognize the modifications of the pulse-wave caused by changes in the elasticity of the artery. It is of much greater diagnostic importance than the pulse-rate. Empty the artery by pressure and roll it to and fro upon the underlying bone. In healthy individuals in early life the artery is felt as a strand of soft elastic tissue. In arteriosclerosis and in those conditions in which the blood-pressure is habitually high, such as chronic nephritis, gout, and lead poisoning, the increased resistance of the artery may be readily recognized. It feels like a whip-cord under the fingers. In advanced arteriosclerosis calcareous deposits in the wall of the artery—atheroma—can be distinctly felt, and in some cases these deposits are so coarse and irregular as to warrant their comparison with a string of wampum. Such arteries are often tortuous. These changes can be best recognized by passing the palpating finger gently along the course of the artery. Important as is the study of the condition of the walls of the peripheral arteries for the

diagnosis of arteriosclerosis, it is nevertheless necessary to call attention to the fact that there are cases of very advanced sclerosis of the aorta and even of the coronaries, and indeed of other deeply situated vessels, in which the superficial arteries upon palpation yield no indication of changes in their walls. To arteriosclerosis, which is often unequally distributed, the radial shows no special liability. It is therefore necessary in suspected cases to examine carefully the superficial arteries in various parts of the body. Increased arterial tension and an accentuated aortic second sound are important signs of arteriosclerosis.

Frequency of the Pulse.—By this term is indicated the number of beats in a minute. It is convenient to count the radial pulse for 15 seconds and multiply the result by 4. If the pulse is irregular or extremely rapid it becomes necessary to count for an entire minute and to repeat the counting in order to avoid error. If after repeated observation wide variations in the frequency are found, the extremes may be recorded. Various devices have been suggested for the counting of very rapid pulses. If regular, every second or third beat may be counted and the result multiplied respectively by 2 or 3; or a dot for each beat may be made with a pencil upon a sheet of paper. These methods are liable to error, and variations in the pulse-frequency uncountable by ordinary methods, that is, exceeding 200, are without clinical importance.

The pulse-frequency is modified by a great variety of physiological influences. The pulse should therefore be counted regularly under similar conditions. When this is impracticable any circumstance liable to influence the frequency should be noted.

Mental excitement in nervous individuals exerts a marked influence upon the frequency of the pulse. The approach of the physician to the bedside or the entrance of the patient to the consulting room is often followed immediately by a rapid increase. It is therefore wise to postpone the taking of the pulse until after some general conversation sufficiently prolonged to enable the patient to regain his equanimity.

The effect of muscular effort in increasing the pulse-frequency is well known. Athletic sports, running, boxing, stair-climbing, and similar effort may be followed by a very rapid pulse-rate which is nevertheless physiological. During convalescence from disease and in feeble and delicate persons slight movements of the body increase the pulse-frequency, which falls again after a period of rest. If, however, the effort be prolonged the return to the normal frequency is delayed.

The pulse-rate is modified by the posture of the body. It rises immediately upon change from the recumbent to the sitting and again from the sitting to the standing position. The frequency attained immediately after these changes falls again in a little time but not to the normal of the previous posture. The pulse-rate for the same individual is relatively higher while each of these positions is maintained. The figures in healthy individuals, in the absence of other modifying conditions, are approximately in the recumbent posture 66, in the sitting 70, in the standing 80 beats per minute.

The pulse-frequency is increased during the digestion of food. Hearty meals and alcoholic beverages render the increase more marked. The diurnal modifications of the pulse bear a definite relation to the periods

of taking food. They occur, however, in those who are fasting and bear some relation to the diurnal variations of the temperature. The pulse's frequency is to some extent modified by respiration, being slightly increased upon inspiration and diminished upon expiration. It is higher after paroxysms of cough. It varies greatly at different periods of life.

Pulse-frequency at Different Ages.—Rollet.

At birth	144-133 per minute
To end of 1st year	143-123 per minute
10th to 15th year	91- 76 per minute
20th to 60th year	73- 69 per minute

Pulse-frequency in Childhood.—Vierordt.

0- 1 year	134 per minute
1- 2 years	110.6 per minute
2- 3 years	108 per minute
3- 4 years	108 per minute
4- 5 years	103 per minute
5- 6 years	98 per minute
6- 7 years	92.1 per minute
7- 8 years	94.9 per minute
8- 9 years	88.8 per minute
9-10 years	91.8 per minute
10-11 years	87.9 per minute
11-12 years	89.7 per minute
12-13 years	87.9 per minute
13-14 years	86.8 per minute

In general terms the frequency declines with advancing years. The pulse in women is about 7 beats per minute more rapid than in men of corresponding age. In large individuals it is slightly slower under similar conditions than in those of smaller size.

Cases are occasionally observed in which the radial pulse is less frequent than the impulse of the heart. This discrepancy arises in consequence of the feebleness of certain contractions of the heart, the pulse-wave not reaching the radials. Under these circumstances the pulse is commonly but not always irregular. In every case of irregularity of the pulse it is desirable to count the contractions of the heart as manifested in the precordial impulse.

In general, departures from the normal pulse-rate, either in the direction of increased or diminished frequency, arise in consequence of derangement of the nervous mechanism of the circulation. Increase may be due to paresis of the pneumogastric or irritation of the sympathetic nerves or the intracardiac ganglia; decrease to irritation of the pneumogastric or paresis of the cardiac sympathetic nerves and ganglia. Derangements of the pulse-rate arise in consequence of causes affecting the myocardium itself.

Increased Frequency—Rapid Heart.—Perhaps the most common cause of an increase in the pulse-rate is the action of the fever-producing toxins. We find it therefore in the febrile infections, the increase in the pulse-frequency bearing a general relation to the elevation of the temperature. The prognosis in severe febrile disease is more favorable where this parallelism is maintained than in those cases in which the pulse-rate is increased out of proportion to the rise of temperature; the very rapid

pulse being the sign of special implication of the heart or vasomotor system. In the acute febrile diseases a pulse-rate of 140–160 in the adult, if maintained for any length of time, is of itself ominous. In children even higher pulse-rates are not uncommon in cases that run a favorable course. The effect of the specific toxins upon the mechanism of the circulation is by no means constant. A knowledge of the variations is of diagnostic importance in doubtful cases. In scarlet fever the pulse-rate is high—120–160—throughout the whole course of the attack; in diseases to which it bears some resemblance, such as angina tonsillaris, diphtheria, rubella, and measles, the pulse-rate of the period of invasion is slower. The pulse-rate in acute miliary tuberculosis and in septicopyæmic conditions is high, out of proportion to the temperature. In malignant endocarditis the pulse is rapid both during the febrile paroxysms and in their intervals. In puerperal sepsis a high pulse-rate is more constant than elevation of temperature. Increased pulse-frequency is common in the early stages of phthisis and usually persists throughout the whole course of the disease, alike in afebrile periods and when the temperature is moderate or excessive.

On the other hand the pulse-frequency of enteric fever is low in proportion to the temperature. In cases of average severity it frequently does not exceed 100–110 with a temperature range during the fastigium of 102° F. (38.9° C.) A.M. to 104° F. (40° C.) P.M. This fact is not without importance in the differential diagnosis between enteric fever and septic infections, the so-called typhoid form of malignant endocarditis and acute miliary tuberculosis. A very rapid pulse in enteric fever is usually the sign of an inflammatory complication or secondary infection.

A frequent pulse occurs in acute affections of the heart, endocarditis, pericarditis, and myocarditis, and in chronic valvular disease in the stage of failure of compensation. Increased pulse-frequency after slight exertion occurs in most forms of chronic myocarditis, in general muscular asthenia, in anæmia, during convalescence from acute diseases, and in conditions of the neighboring organs which subject the heart to abnormal pressure, as pleural effusion, thoracic aneurism, massive enlargement of the liver and spleen, tympany, and ascites. The frequency of the pulse is increased in cardiac palpitation from any cause.

The pulse-frequency is greatly increased in many nervous diseases. A rapid pulse with subnormal temperature is characteristic of shock and collapse. Acceleration of the pulse is a constant symptom of exophthalmic goitre; during the paroxysms of palpitation the pulse is often uncountable. In neurasthenia, Addison's disease, the primary and secondary anæmias, arthritis deformans, and locomotor ataxia the pulse-frequency is likewise habitually increased. In these conditions the rapidity of the pulse may be continuous or show itself only after moderate exertion. In general terms it is proportionate to the severity of the disease. Pain often causes increase in the pulse-rate. Exceptionally slowness of the pulse occurs in connection with very intense pain. In either case the derangement is reflex.

Excesses in alcohol, tobacco, coffee and tea, disorders of digestion, lack of sleep, other exhausting influences, and lowered blood-pressure not rarely produce abnormal pulse-frequency. Certain drugs, as atropine, have the same effect.

Tachycardia—Pycnocardia—Heart Hurry.—The extreme rapidity which follows violent exercise or fright may persist for days or weeks; the rate may reach 160–220. The condition may occur as a pure neurosis. Palpitation and dyspnœa are not always present. The patient is often able to attend to his ordinary duties. Tachycardia is one of the symptoms of the neurasthenic at the menopause and has been attributed to reflex irritation from ovarian or uterine disease. This symptom may be due to lesions such as a tumor or clot in or about the medulla or pressure upon the pneumogastrics.

Paroxysmal tachycardia is a neurosis characterized by attacks of greatly increased action of the heart occurring at irregular intervals and without obvious cause. The attacks usually begin abruptly and are of varying duration, frequently not exceeding an hour or two. The pulse-rate exceeds 200 and is sometimes uncountable. Subjective symptoms may be absent. In many of the cases there is much distress and oppression.

Diminished Frequency—Slow Heart.—In many cases the normal pulse-rate does not exceed 60. In some individuals the pulse may be slow under conditions in which in others it is rapid. This is often the case during the period of convalescence from pneumonia, enteric fever, rheumatic fever, and diphtheria. The pulse is slow while the patient is at rest but is accelerated by slight exertion. It is the slow pulse of exhaustion and occurs in young persons and at the close of uncomplicated cases. Transient slowing of the pulse is a postcritical symptom in certain febrile diseases, as pneumonia. If the pulse-frequency remains high during an abrupt fall of temperature in the course of croupous pneumonia, pseudo-crisis is to be thought of. Slow pulse is encountered in chronic gastritis and ulcer and cancer of the stomach. It occurs in emphysema but is not common in other affections of the respiratory system. It is not rare in aortic stenosis but is infrequent in other valvular diseases of the heart. It is an occasional but by no means constant symptom in chronic myocarditis. Toxic agents, as lead, alcohol, tobacco, coffee, digitalis, and opium, produce slowing of the pulse, and it occurs in some cases of primary and secondary anæmia, diabetes, and myxœdema, especially while the patient is at rest.

Bradycardia—Brachycardia.—The pulse-rate falls as low as 40 and may be persistently slow. It is important to see that the arterial pulse corresponds in frequency with the cardiac contractions. Bradycardia may be physiological or pathological. In rare instances it is a peculiarity of normal individuals. During labor, whether premature or at term, the pulse may fall to 40 or below it. Slow pulse is one of the symptoms of hunger and exhaustion. Cachectic individuals have usually not only subnormal temperature but also a slow pulse-rate. Slowing of the pulse occurs in gall-stone colic, in renal and hepatic colic, and in lead colic. It is associated with acute but not necessarily with chronic jaundice. Either the circulatory mechanism becomes habituated to the bile intoxication or the bile salts are diminished in amount. Bradycardia occasionally occurs in disease of the genito-urinary tract, especially in nephritis and in uræmia. It is of special diagnostic importance in acute cerebral disease associated with intracranial pressure. It occurs in various forms of meningitis,

especially tuberculous meningitis, in which considerable elevation of temperature is sometimes associated with a slow pulse. Chronic cerebral compression, such as results from tumor or hydrocephalus, is not attended with bradycardia except during acute exacerbations. Apoplexy, the postepileptic state, disease of the medulla and diseases and injuries of the cervical cord may be associated with a very slow pulse. Bradycardia occurs in general paresis, mania, and melancholia. It constitutes the essential sign of heart block. A very slow pulse is occasionally associated with shock and may follow the rapid evacuation of large peritoneal or pleuritic effusion.

Rhythm.—Under normal conditions the pulse is regular or rhythmic, that is to say, the individual pulse-waves are of like volume and follow one another at equal intervals of time. Physiological derangements of rhythm are slight and transient and occur under those physiological conditions which are attended by changes in the pulse-frequency. Marked disturbances of rhythm—*arrhythmia*—are always pathological and have their source either in functional derangements of the heart or demonstrable lesions of that organ.

The causes of the various disturbances of rhythm are, (a) psychic or emotional, (b) organic cerebral disease, as endarteritis, hemorrhage, concussion, or compression, (c) reflex, such as produce the cardiac irregularity in gastro-intestinal derangements and diseases of the liver, kidneys, or genito-urinary organs, (d) toxic, the common agents being tea, coffee, tobacco, and alcohol, and finally (e) changes in the heart itself.

Arrhythmia. The following types are to be considered:

1. **Respiratory arrhythmia: Sinus Irregularity of Mackenzie; Pulsus Respiratorius.**—Variation in the length of the diastolic period is the chief characteristic, the systolic period remaining constant. The normal inspiratory increase and expiratory decrease of the cardiac action are exaggerated. It is easily recognized, pulse beats of equal strength but frequency continuously changing with the phases of the respiration being recognized by the finger and upon auscultation. With marked slowing of the inspiration, the pulse shows this irregularity with great constancy. This form of arrhythmia is of vagus origin and is attributed to an exaggeration of the normal respiratory reflex. Jugular tracings show that the right auricle and ventricle contract with the same irregular rhythm as the radial pulse. It has been observed in infants, healthy adolescents and less frequently in healthy adults and is not uncommon in the convalescence from the acute febrile infections, in cerebral disease, especially tuberculous meningitis, neurasthenia and anæmic states. It is of no great diagnostic significance.

2. **Extrasystole; Pulsus Extrasystolicus.**—According to Mackenzie this term should be limited to "premature contractions of the auricle or ventricle in response to a stimulus from some abnormal point of the heart, but when otherwise the fundamental or sinus rhythm of the heart is maintained." As the primary automatic stimulus for contraction arises normally in the remains of the sinus venosus at the mouths of the great veins—sino-auricular node—and since this primitive cardiac tube is represented in the auricle—atrio-ventricular node—and in the bundle of His and its extension to the ventricles, and each section of the heart is capable of independent automatism, the following forms of extrasystole occur:

a. Ventricular Extrasystole.—The origin of the extrasystole is assumed to be in the atrio-ventricular bundle beyond the atrio-ventricular node. The ventricle contracts in advance of its normal time. This contraction is followed by the usual refractory period during which the ventricle fails to react to the normal stimulus from the sino-auricular node with the occurrence of a compensatory pause which is longer than the normal diastolic pause by so much as to make up the loss in duration of the preceding diastole and is followed by a forcible postcompensatory systole in response to the succeeding rhythmic stimulus from the sino-auricular node. It is characteristic of the ventricular extrasystole that the compensatory pause plus the diastole preceding the extrasystole = two normal diastoles.

b. Auricular Extrasystole.—The stimulus arises in the remains of the primitive cardiac tube incorporated in the auricle below the level of the sinus. The premature auricular contraction is followed by ventricular contraction and a long pause after the extrasystole due to the fact that the normal rhythmic stimulus from the sinus occurs during the refractory period of the auricle. The auricle and ventricle are quiescent until the occurrence of the next sinus stimulus. In auricular extrasystole the pause is not usually fully compensatory. The sounds of the heart and the radial pulse do not differ from those of ventricular extrasystole from which auricular extrasystole can only be differentiated by simultaneous tracings of the jugular pulse.

c. Auricular Ventricular Extrasystoles; Nodal Extrasystoles.—This form of extrasystole is attributed by Mackenzie to an abnormal stimulus originating in the auriculo-ventricular node of Tawara and causing premature and simultaneous contractions of auricles and ventricles. Other observers regard it as a ventricular extrasystole in which the stimulus has passed back and prematurely stimulated the auricle—"a retrograde auricular systole."

Extrasystoles constitute the most common causes of arrhythmia. They may occur at regular or irregular intervals, during more or less prolonged periods or continuously, and these three forms of extrasystole may be present in varying combination or in association with other forms of arrhythmia. They are attributed to an undue excitability of the remains of the primitive cardiac tick. Most patients experience no subjective symptoms in connection with the occurrence of extrasystoles. When, however, their attention is called to the irregularity, they are often unnecessarily annoyed by various precordial sensations described as "fluttering," "heart dropping" or the "strong beat" which often follows the long pause. Extrasystoles are of common occurrence in neurotic individuals under various circumstances, especially excitement or exertion, in advanced life, at puberty, the climacteric and during pregnancy; in habitual over-indulgence in tobacco, tea, coffee or alcohol; in the convalescence from the acute febrile infections and in arterio- and cardio-sclerosis. They are essentially of minor clinical importance but the conditions of which they are symptomatic are often grave. Pulsus bigeminus is that form of irregularity in which every second beat is an extrasystole and is usually smaller than the normal beat. The smaller beat is invariably followed by a pause longer than the pause preceding it. The extrasystole may appear after every 1, 2, 3, 4 or

more normal beats and on account of the long compensatory pause is spoken of as an intermission or the "dropping of a beat."

3. **Nodal Rhythm—Continuous Irregularity; Rhythm of Auricular Fibrillation—Ventricular Rhythm; Pulsus Irregularis Perpetuus.**—According to Mackenzie the automatic impulse starts at the auriculo-ventricular node and the auricles and ventricles contract nearly at the same time; according to Cushny the arrhythmia is due to fibrillation of the auricle which ceases to contract as a whole. The condition is characterized by extreme and continuous irregularity of the action of the heart—perpetual irregularity; delirium cordis—by the absence of the sinus rhythm, the presence of a ventricular or positive venous pulse and the absence of an auricular wave in the jugular tracing. The nodal rhythm is present in the majority of cases of severe heart failure and in a great many the immediate breakdown is directly attributed to the inception by the heart of this abnormal rhythm. (Mackenzie.) The heart's action may be (a) not greatly increased and the evidences of cardiac insufficiency may not be marked for a long time, or (b) it may be greatly increased. Sensations of fluttering in the left chest and rapidly developing symptoms of cardiac insufficiency occur. The pulse is small, rapid and except when extremely rapid very irregular in tachycardia; and (c) the nodal rhythm is transient and recurrent; see Paroxysmal Tachycardia. The significance of the nodal rhythm is grave. Improvement in the underlying cardiac condition may occur under rest and the administration of digitalis, but it is transient and exertion is almost always followed by a recurrence of the symptoms.

4. **Partial or Complete Heart Block; Ventricular Rhythm; Pulsus Transmissionus.**—This form of arrhythmia is attributed to partial or complete impairment of the conductivity of the bundle of His. The ventricle fails to contract after the auricular contraction. The stimulus may be delayed or at times prevented from passing over, or finally it may be completely blocked beyond the auricular ventricular node and the contraction of the ventricle then arises in response to a stimulus originating in the functionating remains of the bundle of His—Heart Block; Ventricular Rhythm.

5. **Pulsus Alternans; Exhaustion of Contractility.**—This form of arrhythmia is characterized by an alternating succession of large and small beats, the rate of the heart remaining perfectly regular. It indicates weakness of the heart muscle. The large contraction encroaches upon the period of rest so that contractility has not sufficiently recovered to fully react to the next stimulus, hence the smaller beat due to diminished energy of the heart muscle at the moment. The smaller contraction is in turn followed by a longer period of rest and a correspondingly larger pulse beat. Pulsus alternans is not to be confounded with extrasystole.

Volume.—The volume is the size of the artery under the influence of the pulse-wave. It depends upon the degree of relaxation of the muscular coat. If the expansion is marked the volume is correspondingly great and the pulse is said to be large or full—*pulsus magnus*. If the expansion is slight the pulse is said to be small—*pulsus parvus*. The large pulse is commonly a pulse of low tension. The small pulse varies in tension. If low it is the sign of feeble action of the heart or diminished amount of blood. The

pulse is small and of low tension in valvular disease of the heart with ruptured compensation, and small and usually of good tension in aortic and mitral stenosis.

Celerity.—There is an important distinction between the frequency of the pulse, that is, the number of beats in a minute, and the mode in which the pulse-wave develops under the finger. The pulse is said to be quick—*pulsus celer*—when it is characterized by a wave of rapid ascent and equally rapid recedence. The quick pulse is a pulse of low tension. It is encountered when the peripheral vessels are relaxed, as in the fevers and in various forms of anæmia. Celerity is characteristic of the water-hammer pulse of aortic insufficiency. This pulse occurs also in consequence of the extreme relaxa-

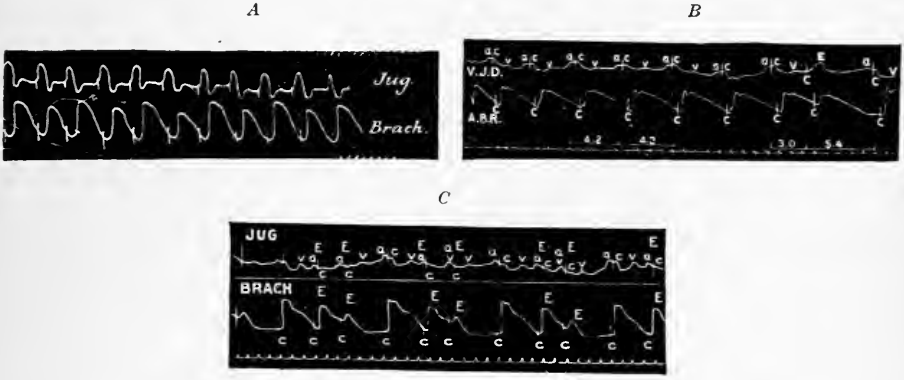


FIG. 198.—*A*, alternating pulse of a case of paroxysmal tachycardia; *B*, pulsus bigeminus. Tracing from jugular vein (V.J.D.) and brachial artery (A.B.R.) showing ventricular extrasystoles; *C*, pulsus trigeminus. Tracing from jugular vein and brachial artery showing two auricular extrasystoles (E,E) after regular systole.

tion of the peripheral arteries in many cases of neurasthenia. In these conditions there is often a visible pulsation in the superficial arteries associated with capillary and, in some instances, with venous pulsation.

The tardy pulse—*pulsus tardus*—is characterized by the gradual rise and equally gradual descent of the pulse-wave. It is a pulse of high tension and is encountered in arteriosclerosis, advanced age, chronic interstitial nephritis, and in some instances during the attacks of angina pectoris. The pulse in aortic stenosis and in arteries peripheral to an aneurism is commonly tardy.

Tension.—This term includes those qualities of the pulse which indicate the arterial blood-pressure. On the one hand the adjectives hard and tense are sometimes used interchangeably in regard to the pulse,—*pulsus durus*,—while on the other hand the adjective soft is used synonymously with relaxed—*pulsus mollis*. The clinician must, however, be constantly on his guard against confounding rigidity of the arterial wall with intra-arterial tension or blood-pressure. It is important also to distinguish between the tension corresponding to the ventricular systole and that corresponding to the ventricular diastole.

High tension occurs in chronic interstitial nephritis, gout, lead poisoning, and in the diabetes of advanced age. The pulse is small and tense in the early stages of acute peritonitis. The pulse tension is increased in pregnancy and in some forms of anæmia.

The pulse of low tension is soft and compressible. It is a sign of cardiac and general asthenia and occurs in all forms of depression and exhaustion. The pulse of obese persons is very often of low tension. Temporary diminution of arterial tension may follow hot drinks, alcoholic beverages, the hot bath, and accompany the period of reaction following great physical exertion or mental excitement. The statement is very often made that arterial tension increases with age. The clinician will do well to recognize the distinction between histological changes in the arterial walls and increase of intra-arterial pressure. When the diastolic pressure is relatively high and the artery remains well filled between the beats, the pulse is said to be full—*pulsus plenus.* When the pulse-wave is very full and quick and the vessels are soft and compressible, the pulse is sometimes spoken of as gaseous. When the artery is collapsed between the beats, the pulse is said to be empty—*pulsus vacuus, vel inanis.* When the pulse-wave is very small and the artery relaxed, the pulse is described as thready, running, or undulatory.

The blood-pressure—arterial tension—may be estimated by the fingers; but more exact and definite measurements are made by various forms of sphygmomanometers.

Dicrotism.—The occurrence of a secondary pulse-wave in each arterial beat is commonly shown in normal sphygmographic tracings. It is recognized by the finger only when fairly well marked. The conditions which favor dicrotism are: diminished arterial tension, relaxed capillaries,—both of which are due to diminished vasomotor tonicity,—a sudden forcible ventricular systole, and relaxation of the arterial walls. Dicrotism may be occasionally recognized by the finger in persons apparently in good health. Such individuals usually manifest a high degree of vasomotor instability, are easily fatigued and bear acute illness badly. The clinical condition in which dicrotism is most marked is fever. It is usually well developed in enteric fever from the beginning of the second week.

OTHER ANOMALIES OF THE PULSE.

The pulse should be examined not only in both radials but, under certain circumstances, in the superficial arteries elsewhere. Retardation, smallness, feebleness, or obliteration of the pulse on one side of the body or locally may be caused by deviations from normal anatomical standards or by traumatism, embolism, thrombosis, tumor pressure, and aneurism.

If the pulse be relatively feeble or small, or if it be absent *in the right radial,* it may indicate an aneurism of the ascending aorta or innominate; *in the left radial,* an aneurism of the transverse or descending portion of the arch; *in a radial of either side* it may indicate the presence on the same side of embolism, thrombosis, aneurism of the subclavian, axillary, or brachial arteries, cervical or axillary tumors exerting pressure upon the vessel, and if slight in degree may be suggestive of pneumothorax or large pleural effusion; *in one femoral, popliteal, or posterior tibial artery* the interference of the circulation may be due to aneurism, tumor pressure, embolism, or thrombosis; *in these vessels on both sides,* to abdominal aneurism or congenital obliteration of the aorta. Absence of pulse in the femorals is an occasional sign of aneurism of the abdominal aorta.

The Pulse in Different Conditions of the Heart—Myocarditis.—In the various forms of sclerosis the pulse is usually feeble. It is sometimes, but not invariably, irregular. It is commonly slow, and not infrequently bradycardia is present, the pulse falling as low as 30 or 40 per minute. In fatty heart the pulse may show the same characteristic. Extreme fatty changes occur, however, without modification of the pulse, which may remain regular and of moderate strength.

Mitral Stenosis.—In the early stages the frequency of the pulse is not increased. It is small and rather tardy, the artery not well filled, the successive beats irregular in time and volume.

Mitral Incompetence.—The modifications depend upon the extent of the lesion and the condition of the left ventricle. The frequency is increased, the volume and tension are diminished, the rhythm usually, but by no means constantly, irregular.

Aortic Stenosis.—In uncomplicated cases the pulse is slow, its volume diminished, its tension maintained, the artery being well filled during the ventricular diastole. The pulse of aortic stenosis is usually regular.

Aortic Incompetence.—The large mass of blood propelled into the aorta by the dilated and hypertrophied left ventricle causes sudden distention of the arterial system, which is followed by an equally sudden collapse resulting from the failure of the base of support to the column of blood normally supplied by the aortic valves; characteristic and striking changes in the arterial pulse result, and are manifested in all the superficial arteries. The pulse is increased in frequency and usually regular. The artery is suddenly distended, the pulse being quick, jerking, visible, and abruptly receding. The pulse is locomotor, that is to say, the visible arteries are elongated and their curves accentuated. The jerking, visible, and collapsible character of the pulse, as observed in the arm and wrist, is intensified by elevating the member above the head. Very often there is pulsation at the root of the neck, and in well-developed cases there is distinct pulsation of the tissues of the hands and feet—capillary pulse—which may be felt by gently grasping the hand or foot, placing the thumb upon the palmar or plantar surface and the fingers upon the dorsal surface. With the ophthalmoscope pulsation of the retinal arteries may be seen. Capillary and the so-called penetrating venous pulsation also occur.

The pulse of aortic incompetence is frequently called the Corrigan pulse, after Sir Dominic Corrigan, who first systematically studied and described its peculiarities.

Arteriosclerosis.—Modifications of the pulse in arteriosclerosis depend upon the peripheral resistance and the force of the ventricular systole. Sclerosis and high tension are usually associated. The pulse-wave is tardy, sustained, and subsides slowly, the vessel remaining full between the beats—diastolic tension. Pressure of the finger does not readily obliterate the pulse. An effort must be made to discriminate between the firmness due to intra-arterial tension and that due to thickening of the arterial wall. If, when the pulse-wave is arrested by the pressure of the finger, the artery can be felt beyond the point of compression, its walls are thickened.

CAPILLARY PULSE.

Normally the pulse-waves penetrate to the smallest arteries but are lost in the capillaries. Under certain circumstances, however, the pulse is manifest in the capillaries and shows itself upon inspection as a pulsatile flushing and fading of the surface. Conditions which especially favor the development of the capillary pulse are relaxation of the peripheral circulation and rapid discharge of a large amount of blood from the ventricle into the arterial system—*pulsus celer*. Capillary pulse is occasionally seen in areas of local hyperæmia and inflammation, as in whitlow, and patients often recognize this increased pulsation in the throbbing character of the pain. Of greater interest is the capillary pulse of aortic incompetence. It is seen in the pulsatile changing in the color intensity of the nail-bed—*subungual pulse*—a phenomenon which becomes more marked when the nail is slightly pressed near its edge so that the underlying tissue is momentarily pale. The border line between the pink and white advances and recedes with each cardiac revolution. The capillary pulse can be distinguished in the ear, lips, cheek, and especially distinctly upon the forehead at a point where it is reddened by light rubbing. The capillary pulse shows clearly through a piece of glass pressed upon the mucous surface of the everted lip. It is, however, not confined to aortic regurgitation, but occurs also in pyrexia, chlorosis, and other forms of anæmia, neurasthenia, and exophthalmic goitre. The capillary pulse of aortic incompetency is more marked in stages in which the compensation is good and disappears upon the failure of compensation.

VENOUS PULSE.

The modifications of intrathoracic pressure caused by respiration are of importance in connection with the venous circulation. Inspiration hastens, expiration retards the flow of blood in the veins. These modifications are not seen upon ordinary quiet breathing. On the other hand, in forced respiration and dyspnœa there is expiratory venous distention and inspiratory venous collapse, best seen in the veins of the neck, especially when they have been enlarged by previous congestion and are therefore more distinctly visible. Even more marked is the influence of the variations of intrathoracic pressure by violent cough and prolonged muscular effort. In individuals in which these paroxysms of intravenous pressure recur through long periods, as in those who suffer from chronic paroxysmal cough, the veins, especially the jugular, become permanently enlarged, so that during the paroxysms the patient is not only cyanosed but manifests a distinct distention of the vessels at the root of the neck. The jugular bulb sometimes appears as a small, sausage-like swelling in the region of the insertions of the sternocleidomastoid muscle.

More rarely distention of the veins takes place during inspiration; retraction upon expiration. This reversal of the ordinary conditions is the result of mechanical interference by pressure or traction upon the great veins within the thorax, such as occurs in chronic mediastinitis, mediastinal tumors or a large pericardial or pleural effusion,—conditions in which Kussmaul's pulsus paradoxus is frequently observed.

The Forms of Venous Pulse.—Pulsation in the veins is due to the movements of the heart and has the cardiac rhythm. A pulsation communicated by the underlying carotids is sometimes seen in the external jugular veins. This is the so-called false venous pulse. The distinction between this and true venous pulsation is usually unattended with difficulty. The more extended superficial pulsation due to the greater width of the vein and the peculiar, prolonged, undulatory movement which is characteristic of the low intravenous tension are of importance. Upon light palpation the pulse is feeble and compressible and in strong contrast to that elicited upon palpation of the underlying artery. Upon compression of the vein the peripheral pulsation continues or may be increased in consequence of the distention; upon compression of the artery at the root of the neck the pulsation wholly disappears.

True Venous Pulse.—Three forms are encountered: the physiological or negative venous pulse, the regurgitant or positive venous pulse, and the penetrating or positive centripetal venous pulse.

(a) Negative Venous Pulse.—This form of pulsation in the veins is called normal or physiological because it is constantly seen in the exposed veins of animals and frequently in the jugulars of human beings in health. It is not observed in all persons, simply because the jugular veins in many individuals are difficult or impossible to distinguish. It is very obvious upon inspection in those persons in whom the veins are distended and plainly visible. The physiological venous pulse is readily distinguished from positive or regurgitant venous pulsation by compression of the vein with the finger. The pulsation peripheral to the point of compression ceases, and that central to it likewise disappears or becomes much fainter. The diminution or complete disappearance in the latter case makes it evident that the pulse-waves are not transmitted to the blood in the veins by the cardiac systole. It is thus apparent that the continuous blood stream from the veins is rhythmically restrained and hastened by the action of the heart. The negative venous pulse is observed in the external and internal jugulars. It is presystolic in time. The collapse of the vein at the time of the ventricular systole is attributed to the negative intrathoracic pressure caused by the diminution in the size of the heart at that moment in its revolution. During the ventricular diastole the aspiration influence ceases and the blood accumulates in the veins. It may be urged against this statement, however, that the venous pulse, central to the point of compression, though always reduced, does not in all cases wholly disappear; but the external jugular cannot be entirely emptied of blood, as there are tributary veins central to the point of compression. In some cases this form of venous pulsation can be still further reduced by simultaneous compression of the subclavian. In order to determine the time of the venous pulse, which is diastolic,—presystolic,—it must be compared with the carotid pulse, which is, of course, systolic. The negative venous pulse is without diagnostic significance. A knowledge of it is necessary, however, in order that it may be differentiated from the form about to be described.

(b) Positive or Regurgitant Venous Pulse.—This form of venous pulsation is observed in tricuspid incompetence. During the ventricular systole the blood regurgitates into the right auricle and the pulse-wave is transmitted to the vein. The pulsation is presystolic-systolic rather than

purely systolic, as in the case of the arterial pulse. When the valve in the jugular is competent the pulsation is more marked in the bulb, but it does not always cease at the level of the valve even when competent. The regurgitation is interrupted, but a positive pulse-wave of similar form, though weaker, is induced in the blood which accumulates above the valve. In some instances the closure of the valve under the influence of the regurgitant blood wave gives rise to a sound distinctly audible upon auscultation. In the majority of instances, as a result of the over-distention of the veins, the valve becomes insufficient, so that the positive pulse is equally perceptible over the upper portion of the jugular. The distinction between the positive and negative venous pulse rests upon the correspondence of the former with the carotid pulse and its persistence in the pulsating vein below the point of compression. As a rule, positive venous pulsation is observed only in the jugulars.

This form of pulsation is a sign of tricuspid incompetence. It has, however, been observed in two extremely rare conditions in which the lesions likewise favor the transmission of the systolic pulse-wave to the jugular veins, namely, mitral incompetence with persistent foramen ovale and aneurism of the aorta communicating with the descending vena cava.

The patient should be examined in the recumbent posture and during very quiet breathing. Before making compression in the course of the vein the finger-nail should be placed upon the vein at the root of the neck and lightly drawn upward to empty the vessel. In the absence of regurgitation the vein refills slowly, but if the tricuspid valves be incompetent the vein quickly refills from below and again pulsates.

Pulsation of the Liver.—In advanced cases of tricuspid incompetence the liver becomes enlarged and the hepatic veins dilated and engorged. In this condition the organ pulsates, the regurgitant wave being transmitted through the inferior vena cava. The pulsation may be recognized upon palpation, one hand being placed over the cartilages of the lower ribs to the right of the ensiform cartilage, and the other upon the side at the costal margin. An expansive pulsation of the entire organ can be felt with each cardiac impulse. In marked instances liver pulsation may be made out upon inspection. Pulsation of the liver must be distinguished from the jogging of the organ by a powerfully acting hypertrophied heart. It must also be distinguished from the epigastric pulsation of the abdominal aorta—dynamic pulsation—and from aneurismal pulsation. In very rare cases of aortic regurgitation, with good compensation and no sign of tricuspid incompetence, an arterial liver pulse has been noted, and local pulsation with double murmur has been observed in acute cholangeitis.

(c) Penetrating or Positive Centripetal Venous Pulse.—This rare phenomenon is due to the fact that under certain conditions the pulse-wave is not lost in the capillaries but transmitted through them to the smaller veins. It has the same significance as the capillary pulse and occurs in cases of aortic incompetence or neurasthenia with great vasomotor relaxation. It has been observed in cases in which the capillary pulse has been faintly perceptible or absent altogether. It is associated with quick arterial pulse of large volume and is manifest not in the jugulars but in the small veins of the extremities, and disappears upon compression in the central, not in the peripheral, portion of the compressed vein.

In this connection diastolic collapse of the cervical veins, the so-called Friedreich's sign, may be mentioned. This sign occurs in chronic adhesive pericarditis but is of no great diagnostic value. The collapse of the veins is due to diastolic intrathoracic aspiration. The mechanism is the reverse of that in the physiological venous pulse.

VII.

THE DIGESTIVE SYSTEM: MOUTH; LIPS; TEETH; GUMS; TONGUE.

THE MOUTH.

The most important method of examination is inspection. The patient should be placed in a good light. The illumination is more satisfactory by light reflected from a head mirror. The mouth should be opened widely and, according to the part to be examined, the tongue should be protruded, drawn back, or moved from side to side. The soft palate and pharynx are best seen upon depression of the base of the unprotruded tongue with a spatula or the handle of a spoon. These instruments, if introduced too far, cause gagging. The examination of the posterior wall of the pharynx is facilitated when the patient pronounces the broad *a*, thus elevating the soft palate. In conditions of delirium or unconsciousness and in insane patients the examination of the mouth is often attended with great difficulty. In some instances holding the nose will cause the patient to open his mouth; in others, if necessary, the patient must be anæsthetized. In the case of children the examination is best conducted when the physician and mother or nurse sit viz-a-viz, the body of the child resting upon the knees of the latter, who holds his hands, the head upon the lap of the former, who opens the mouth and depresses the tongue with the spatula. The pharynx is best seen at the beginning of gagging. Palpation by means of the finger is very useful in detecting the presence and location of foreign bodies, the existence of retropharyngeal abscess, and especially adenoid vegetations in the nasopharynx and other similar conditions. This manœuvre, which is very annoying to the patient, must be executed rapidly. In the case of unruly patients or children the danger of being bitten is not to be overlooked. Against this accident a guard or shield may be employed, or the physician may, with the thumb and finger of the free hand, press the cheeks of the patient between the separated molars.

THE LIPS.

The lips are thick and coarse in habitual mouth-breathers, in cretinism, and in myxœdema. They are parted in conditions of great prostration and habitually in idiots and in some forms of insanity. They are pallid in anæmia and, like the nail-beds, early show cyanosis and the variations in its intensity. The lips are apt to be dry in dyspnœa and in obstruction

to the nasal breathing. Dryness of the lips is associated with a diminution or perversion of the oral secretion, as in stomatitis, glossitis, and tonsillitis. The lips and mouth are dry and the latter open in the soporose condition preceding dissolution. There is drooling in dentition, mercurial salivation, diphtheritic paralysis, bulbar palsy, and idiocy. In these conditions the lips are apt to be loose and pendulous. Tremor or twitching of the lips occurs under intense emotion or may be a symptom of nervous disease. Convulsive retraction of the upper lip occasionally occurs as a sign of intense abdominal pain. Of great diagnostic importance is the occasional presence upon the lips of aphthous ulceration, mucous patches, sordes, rhagades—linear clefts or ulcerations at the corners of the mouth—or the scars resulting from them. The last, occurring in young children, are suggestive of hereditary syphilis. Herpes labialis is common in certain individuals in feverish colds, and occurs with such frequency in pneumonia, ague, and cerebrospinal fever as to have diagnostic value. It is very rare in enteric fever. This vesicular eruption develops rapidly upon an inflammatory base as a single lesion or in groups, most commonly upon the outer border of the lip, occasionally on other parts of the face, as the nose, the cheeks, or the ear. These positions are indicated by qualifying adjectives, as *herpes labialis, facialis, nasalis,* and the like. The contents of the vesicles are at first lymphoid, later purulent and scanty. Their efflorescence is attended by annoying burning or itching. They rapidly undergo desiccation with the formation of thick, tightly adherent scabs. The whole process is of short duration.

In paralysis of the seventh nerve the angle of the mouth on the affected side is lowered. In drinking, the liquid is apt to escape. In smiles or laughter the corner of the mouth is immobile and in attempts to show the upper teeth it is not raised. The mouth and lips are drawn toward the sound side. The labial sounds may not be fully formed. It is important to note that the displacement of the angle of the mouth may be due to loss of the teeth upon the opposite side or to retraction as the result of scar formation.

The lips are extremely sensitive and abscesses and acute inflammatory processes are attended with great pain. They are sometimes the seat of carbuncles. They undergo extensive necrosis in *cancrum oris.* The lip may be lacerated in the epileptic convulsion, but this is not common. It may be the seat of angioneurotic œdema or may be greatly swollen in consequence of the bites of insects. The lip is occasionally the seat of the initial lesion of syphilis. It shows more or less extensive superficial necrosis extending out upon the chin or cheeks after the taking of corrosive poisons and especially in carbolic acid poisoning. Epithelioma of the lip is common. It shows itself as an irregularly circular or oval ulcer with a swollen, infiltrated base, usually upon the lower lip, developing from a fissure or wart. At the beginning there are alternations of scab formation and open ulceration. After a time the submaxillary lymphatics become involved.

The differential diagnosis between chancre and epithelioma of the lip is usually unattended with difficulty. The chancre occurs, as a rule, early in life, epithelioma late. In chancre the lymphatics are involved

early; in epithelioma late. Chancre is commonly circumscribed and densely indurated; epithelioma tends to spread and the induration is less dense. Healing of the chancre is progressive, especially under treatment; in epithelioma there is a tendency to extend, with alternations of scab formation and ulceration. In the former, constitutional symptoms and secondary rashes occur.

THE TEETH.

The teeth are of diagnostic interest. The time of their eruption and shedding in children and their state of preservation in adults are to be considered. Dentition and teething are terms used to describe the cutting of the teeth.

The First Dentition.—The temporary or deciduous teeth—the so-called milk teeth—are twenty in number; in each jaw two central incisors, two lateral incisors, two canines, two first molars and two second molars. They appear with considerable regularity as to order and time. Their eruption usually takes place in groups of four.

The first group—the lower and upper central incisors, 6 to 9 months. An interval of 1 to 3 months.

The second group—the upper and lower lateral incisors, 8 to 12 months. An interval of 1 to 3 months.

The third group—the four anterior molars, 12 to 15 months. An interval to the 18th month.

The fourth group—the four canines, 18 to 24 months. An interval of 2 to 3 months.

The fifth group—the four posterior molars, 24 to 30 months.

Healthy children usually have from four to eight teeth before they are a year old, and cut their first molars between a year and a year and a half, the canines before the end of the second year, and should complete dentition by the cutting of the second molars before the middle of the third year. The first teeth are usually the lower central incisors. The upper lateral incisors as a rule appear before the lower; the upper first molars usually precede the lower and not infrequently appear at about the same time with the lower lateral incisors.

Precocious dentition occasionally occurs. It is of no special significance. Delayed dentition occurs as the result of malnutrition either from improper feeding or disease. It is especially common in rickets.

The eruption of the teeth in healthy, well-nourished children commonly takes place without constitutional disturbance. At most transitory loss of appetite, fretfulness, disturbed sleep, a slight rise of temperature, 100–102° F. (37.7–38.8° C.), and derangement of the bowels are observed. In feeble and poorly nourished infants, especially in neurotic families, the perturbations caused by dentition may be more severe, the foregoing symptoms being aggravated and the temperature reaching higher levels, 103–104° F. (39.4–40° C.). The accidental coincidence of gastro-intestinal derangements, tonsillitis, laryngitis, and bronchial catarrh is very common, and the physician must be on his guard not to ascribe to dentition symptoms which are due to other causes. On the other hand there is danger that reflex symptoms due to the irritation of dentition will be

erroneously interpreted. For example, annoying spasmodic cough, without fever, other constitutional disturbance or râles, and manifestly reflex, frequently accompanies the eruption of each group of teeth. Dentition may be the exciting cause of general convulsions in feeble, badly-nourished, rhachitic, or neurotic children. The process rarely causes eclampsia in well-nourished healthy babies. Tension, tumefaction, tenderness of the gums, and the bluish-red hue of deep congestion are indications for the use of the lancet.

The Second Dentition.—The permanent teeth in each jaw consist of two central and two lateral incisors, two canines, four bicuspids, and six molars. Their eruption takes place in the following order:

Anterior molars,	sixth to seventh year.
Central incisors,	seventh to eighth year.
Lateral incisors,	eighth to ninth year.
Anterior bicuspids,	tenth to eleventh year.
Posterior bicuspids,	tenth to eleventh year.
Canines,	eleventh to twelfth year.
Second molars,	twelfth to fourteenth year.
Third molars—wisdom teeth,	eighteenth to twenty-fifth year.

The milk teeth are gradually displaced by the permanent teeth and three additional molars appear on the sides of each jaw, so that the twenty milk teeth are replaced by the full set of thirty-two permanent teeth. The second dentition begins with the eruption of the anterior molars somewhere between the fifth and seventh years. Following these the milk teeth are gradually shed in the order in which they appeared, each tooth being forthwith or shortly replaced by a permanent tooth.

Shape and Structure of the Teeth.—Defects in the teeth are numerous, the most important being abnormalities of form, and especially the deficient development of enamel. In badly-nourished, feeble children the milk teeth are prone to caries.

The developing teeth are influenced by malnutrition, stomatitis, especially that produced by mercury, and constitutional diseases, as syphilis and rickets. The developmental defects show themselves in the permanent teeth. In rickets the teeth may be small and badly formed. As the result of infantile stomatitis the surfaces of the teeth are pitted, owing to deficient formation of enamel; the condition is sometimes improperly spoken of as *erosion*. These changes affect the incisors and canines, which are pitted by areas of default of enamel, and are of a bad color, showing a transverse furrow across all the teeth at the same level; the first permanent molars are also involved. These furrows are attributed, probably correctly, to severe illness in early life and are regarded as analogous to furrows on the nails which occur after serious disease.

Hutchinson Teeth.—In congenital syphilis the teeth are deformed and present appearances regarded by Hutchinson as specific and peculiar. The upper central incisors are affected. They are peg-shaped, short, and narrow, being smaller at the cutting edge than at the base. The enamel is commonly well formed and regularly developed, but the color is more yellow than that of the other teeth. At the edge of the teeth there is a single concave notch of varying depth in which the dentin is exposed. They are called Hutchinson, notched, or screw-driver teeth. These defects

are not constant nor are they pathognomonic of syphilis, as they are sometimes found in other conditions, especially rickets. In the presence of other signs of syphilis—rhagades, keratitis, iritis, and nodes—notched teeth acquire positive diagnostic importance.

Fig. 199.—Hutchinson's teeth.

Caries.—Carious and neglected teeth play a very important part in the causation of derangements of digestion from imperfect mastication, and are themselves not rarely the result of constitutional disturbances. Extensive and rapid dental caries may occur after serious acute disease and in constitutional disorders as rickets and diabetes. It also occurs in pregnancy. The teeth become loose in forms of stomatitis associated with swollen and ulcerated gums, such as are encountered in mercurial ptyalism, scurvy, purpura, and phosphorus poisoning. Receding gums with exposure of the neck of the teeth and their ultimate loss occur from neglected salivary deposits, pyorrhœa alveolaris, and gouty conditions.

Sordes—literally filth—is a term applied to collections of dark brown foul matter upon the teeth and lips in low fevers. It consists of food, epithelial material, and altered blood, and contains micro-organisms in great numbers.

THE GUMS.

The gingival mucous membrane is pale in all forms of anæmia; it is red and spongy when the teeth are carious or ill-kept. A narrow red line along the margin is seen in some cases of tuberculosis, diabetes, and in cachectic states; also in alveolar disease. The gums are red, spongy, and ulcerated as a result of accumulated tartar and gangrenous and mercurial stomatitis. They are swollen, spongy, and bleeding in scurvy.

In lead poisoning a narrow bluish-black line is seen, although not invariably, at the margin of the gums. The color is not uniform, but, being due to lead sulphide deposited in the papillæ of the gums, is seen with the magnifying glass to be stippled. This line may form rapidly after exposure and disappear in the course of a few weeks under treatment, or it may persist for months. It is usually limited in extent. A similar line, due to the deposition of carbon particles, has been observed in miners. Such lines are to be distinguished from the deposits of black matter upon the teeth at the line of their juncture with the gums in untidy persons and smokers who neglect the mouth. The latter disappears upon the use of the tooth-brush, or the two lines may be differentiated by slipping the corner of a piece of writing-paper under the gum. If the pigment material is in the gum it stands out plainly against the white paper; that on the tooth is not seen. It is under certain circumstances also to be distinguished from cyanosis due to general disturbances of the circulation or local inflammatory processes. In cyanosis the discoloration is uniform and more intense at the edges of the gums and disappears under pressure.

THE TONGUE.

Great differences of opinion exist in regard to the value in diagnosis of signs presented by the tongue. To the careful observer an examination of the tongue yields information of diagnostic importance. This organ should be studied with reference to its motility, size, condition of the mucous membrane as regards color, papillæ, dryness, moisture, coating, and the presence or absence of various lesions.

(a) **Motility.**—The manner in which the tongue is protruded upon request is often suggestive. Under ordinary circumstances the movement is commonplace and familiar. In very ill patients the tongue is protruded slowly and incompletely. In the advanced stages of enteric fever the patient protrudes his tongue hesitatingly and does not immediately withdraw it unless requested. In chorea the tongue is thrust out with a peculiar jerk and immediately withdrawn. In well-marked cases it is impossible for the patient to keep it out for any length of time. Spasm of the muscles of mastication renders it impossible to protrude the tongue. The spasm may be tonic or clonic; in rare instances it occurs as an independent affection. It is usually part of general convulsive disease. In the tonic form the jaws are held forcibly together—lockjaw. The masseter and temporal muscles are tense and hard and the spasm is frequently attended with pain. It is an early and prominent symptom in tetanus and occurs also in tetany. There is tonic spasm of the jaw muscles in trismus neonatorum and strychnia poisoning and sometimes in hysteria and epilepsy. Trismus may follow exposure to cold or occur as the result of reflex irritation in diseases of the mouth, teeth or jaw or of irritative lesion in the region of the motor nucleus of the fifth nerve. Clonic spasm of the muscles of the jaw is seen in the chattering of the teeth which occurs after exposure to cold, in some conditions of mental excitement, and during a chill. Its rare occurrence as a substantive affection has been noted. Pain and swelling of the tissues about the angle of the jaw, such as attend disease of the bones, mumps, suppurative tonsillitis, and trichinosis involving the masticatory muscles, may prevent the opening of the mouth and protrusion of the tongue.

General tremor of the tongue occurs in alcoholism and in conditions of asthenia. Tremor and fibrillary contractions are observed in patients presenting bulbar symptoms with atrophy of the tongue and may be especially pronounced in progressive bulbar atrophy. Fibrillary contractions are occasionally seen in healthy individuals.

Paralysis of the tongue results from disease of the hypoglossal nerves. When one nerve is involved the base of the tongue is slightly higher upon the paralyzed side, and motion within the mouth toward that side is impaired. When the tongue is protruded it deviates to the paralyzed side, being pushed by the geniohyoglossus on the normal side; there is slight difficulty in chewing and swallowing. When both hypoglossal nerves are involved the tongue cannot be moved within the mouth and cannot be protruded; mastication and articulation are greatly impaired. Palsy of the tongue from nuclear disease is usually associated with a similar condition of the lips, pharynx, and larynx. The power of protruding the tongue

is impaired in paresis, diphtheritic palsy, progressive muscular atrophy, and some forms of hemiplegia. Slight deviation toward the paralyzed side may occur in cases of hemiplegia in which the face is affected. When the fibres of the hypoglossal are involved within the medulla after leaving their nuclei, there may be paralysis of the tongue on one side and paralysis of the limbs on the other, and the tongue when protruded deviates toward its sound side. Other causes of nuclear or infranuclear lesions of the hypoglossal are lead poisoning, basal meningitis, and tumors of the base.

Spasm of the tongue is very rare. It may be unilateral or bilateral —tonic or clonic. It is usually one of the manifestations of some other convulsive disease, as spasm of the facial muscles, tetanus, epilepsy, or chorea. Tonic spasm may occur in hysteria and as the result of reflex irritation of the fifth nerve. The tongue is contracted and rigid. Clonic spasm is much more common. Spasm of the lingual muscles occurs in stuttering. It is an occasional symptom in disseminated sclerosis, general paresis, and melancholia. There are cases of paroxysmal clonic spasm in which the tongue is thrust out and drawn in as often as forty or fifty times a minute. In this affection the spasm is usually bilateral; the attacks may occur during sleep.

The frænum of the tongue may be abnormally short—a congenital defect which, by limiting the movements of the tongue, interferes with nursing in the new-born and with articulation later.

(b) **Size of the Tongue.**—Variations in the size of the tongue are of diagnostic importance. The tongue is slightly enlarged and flabby in various conditions of ill health and especially in chronic gastritis, forms of anæmia, scurvy, and typhus fever. Under these circumstances the edges are indented by the teeth.

Enlargement of the tongue, or *macroglossia*, is usually congenital but may occur in later life. In the congenital form the tongue and very often the lips are greatly enlarged by an increase in all the tissue elements, an increase in the fibrous tissue alone, or from the development of tumor-like masses—true lymphangioma. The organ may become so large that it projects beyond the teeth, in some cases attaining twice its normal size. The surface is dry, fissured, or ulcerated from contact with the teeth, and deformity of the bony structures results from pressure. The lymph-vessels are dilated and in some instances there are actual cysts.

Great enlargement takes place in acute inflammation of the tongue, such as glossitis, inflamed ranula, erysipelas, angina Ludovici. The tongue is frequently much enlarged in actinomycosis. One side only may be involved in the inflammatory process—*hemiglossitis*. The tongue is somewhat enlarged in acromegaly and myxœdema. Localized swelling may be caused by tumors, as gumma or carcinoma. The tongue in rare instances becomes cyanosed and œdematous from obstruction to the return of the venous blood.

Diminution in the size of the tongue may be the result of a temporary shrinking or of atrophy. The tongue may be uniformly diminished in size after hemorrhage, during convalescence from enteric fever, or in conditions of advanced emaciation.

Atrophy of the tongue is the result of disease in the path of the hypoglossal nerve. If the lesion be supranuclear there is no wasting of the tongue. There may be some degree of paralysis. Ordinarily this condition constitutes an element of hemiplegia. In nuclear or infranuclear paralysis the tongue is atrophied on one or both sides according as the lesion is unilateral or bilateral. The muscular tissue is alone affected, ordinary sensation and taste remaining practically normal. The reaction of degeneration is present in the wasted half of the tongue.

Facial hemiatrophy is usually associated with hemiatrophy of the tongue on the same side. Local diminution in the size of the tongue may follow the resorption of a gumma or extensive scar formation following a deep ulcer.

(c) **Mucous Membrane.**—The color of the organ itself is to be distinguished from the coating. The tongue is pale in anæmia; red in inflammation, as glossitis and stomatitis, and in the infectious diseases, as measles, scarlatina, and enteric fever; dark red in conditions of prostration; bluish in cyanosis; yellow in jaundice. It is stained various colors by ingested articles—red or purple by fruits or wine, black by iron, bismuth, or charcoal, yellow by rhubarb, tobacco, or licorice root, brown by chocolate and opium. Ingestion of corrosive substances may give rise to staining with superficial necrosis. Ammonia, corrosive sublimate, sulphuric, carbolic, and oxalic acids turn the tongue white; hydrochloric, nitric, chromic, and picric acids yellow; the caustic alkalies turn it red. Local discoloration of the tongue is caused by telangiectatic patches, purpura, ecchymoses, and infarcts. Patches of pigmentation may mark the site of healed glossitis or occur as manifestations of Addison's disease. In the latter condition the color is bluish- or grayish-black and the areas of pigmentation are associated with similar areas of pigmentation upon the buccal mucous membrane and the lips. The "black tongue" or *nigrities* is a rare affection of parasitic origin. It is characterized by irregular areas of blackish-brown or black color, with enlargement of the papillæ, which occupy the middle of the dorsum of the tongue. The discoloration begins as a small spot and extends; after a time desquamation occurs which goes on slowly. The condition may become chronic. It is to be distinguished from staining of the tongue caused by iron, bismuth, and the like, and from purposeful discoloration in malingering.

Moisture.—The normal tongue owes its moisture to the buccal secretions and saliva. A physiological increase of these secretions occurs in hunger and is excited by the sight or odor of food. Such an increase is also promoted by sapid and stimulating substances and by mastication. It is called salivation or ptyalism. It occurs during dentition, menstruation in some instances, often during pregnancy—usually in the early months but sometimes throughout the whole period. Jaborandi and its alkaloid pilocarpine, muscarine, tobacco, mercury, gold, copper, and the iodine compounds excite an increased flow of saliva. A pathological increase of saliva occurs in forms of glossitis and stomatitis, especially that induced by mercury, sometimes in the fevers, in the epileptic paroxysm, and in some forms of idiocy and nervous disease. It has been observed in disease of the pancreas.

Xerostomia—dry mouth—is a condition characterized by arrest of the salivary and buccal secretions. The condition is rare. It was first described by Jonathan Hutchinson. The tongue is red, dry, and fissured; the buccal mucous membrane is smooth and dry. Movements of the parts involved in articulation, mastication, and deglutition are attended with difficulty. In some cases the dryness extends to the nostrils and eyes and is accompanied by distressing itching. Slight enlargement of the salivary glands has been observed but is not constant; most of the cases occur in women of neurotic constitution. In a case under my observation in a woman aged thirty this condition developed during the convalescence from an attack of epidemic influenza. It has been suggested that the disease is due to involvement of a hypothetical centre controlling the salivary and buccal secretions.

Dryness of the tongue occurs in mouth-breathing, with thirst, after violent exertion, in febrile and septic states, conditions of profound prostration, and as the result of loss of fluid in diabetes mellitus and insipidus. It is an important symptom of atropine poisoning, and attends facial paralysis. Dryness of the tongue occurs under other conditions attended with extreme loss of fluid from the body, as in hemorrhage and cholera.

The papillæ of the tongue are often swollen, giving it a warty, granular appearance. This condition is seen in catarrhal and other forms of stomatitis, in some forms of chronic gastritis, and sometimes in the acute febrile infections. The enlarged fungiform papillæ of the tongue in scarlet fever have given rise to the unfortunate term "strawberry tongue," which by some teachers is understood to mean a tongue covered with a white fur through which the tip of the papillæ show, and by others to mean the rough bright red tongue which follows the separation of the coating. The latter is sometimes called the "raspberry tongue." In conditions of prostration, such as attend the later stages of infections or sepsis, and in some constitutional diseases, as diabetes, the tongue sheds its epithelium and the papillæ undergo atrophy. This condition is usually attended with dryness and glossing of the surface. The papillæ at the border of the tongue are sometimes greatly enlarged in gouty individuals. Patients are occasionally alarmed upon the discovery of the large circumvallate papillæ at the root of the tongue and hesitatingly accept the assurance that they are normal.

Coating of the Tongue.—This subject involves a consideration also of the general condition of the mucous membrane as regards color, dryness and moisture, and the condition of the papillæ. The presence or absence of coating is determined by local and constitutional conditions. It does not follow, as is very often assumed, that the condition of the tongue is directly dependent upon the condition of the mucous membrane of the stomach. On the contrary the diagnostic significance of coated tongue will be best understood by the clinician who realizes the fact, of which there is abundant clinical demonstration, that the condition of the tongue as regards coating and allied phenomena is largely dependent upon constitutional influences which are likewise exerted upon other mucous surfaces. Coating of the tongue occurs in many morbid conditions, especially dyspeptic states and in fevers, and is usually associated with loss

of appetite; yet there are healthy individuals with good appetite whose tongue is constantly furred. A coated tongue is present in acute and chronic gastric catarrh, while on the other hand gastric ulcer is very often accompanied by a clean tongue and good appetite. The coating or fur is composed of accumulated epithelium and food detritus and contains great numbers of micro-organisms. The immediate cause of the extraordinary proliferation and accumulation of epithelial elements is not well understood. That the absence of coating is not merely dependent upon mechanical conditions associated with drinking and the ingestion of food is clearly shown by clinical experience. The coating of the tongue like its mucous membrane is very often stained by articles of food and drink or by drugs.

Coating of the Tongue in Local and General Conditions.—(a) LOCALIZED COATING of the tongue results from the irritation of a tooth and surrounds traumatic and other circumscribed lesions.

(b) UNILATERAL COATING of the tongue is sometimes seen in trifacial neuralgia involving the infra-orbital branch. It may occur also in unilateral palsy of the tongue.

(c) A UNIFORM THIN, WHITISH COATING is habitual to many persons in health, especially mouth-breathers, smokers, and those who are troubled by subacute catarrhal processes involving the pharynx and stomach. It occurs also in constitutional disturbances attended by slight fever.

(d) A THICKISH, PASTY, YELLOW-WHITE FUR is common in those addicted to excesses at table or in tobacco or alcohol. It is attended with a disagreeable taste. On rising it usually involves the greater portion of the dorsum of the tongue, but disappears in part or wholly during the day. In many persons this coating remains upon the back part of the tongue continuously. Its disappearance is to some extent due to movements of the tongue, friction against the teeth, the mechanical effects of food and drink, and increased flow of salivary and buccal secretions. A slightly enlarged, flabby, indented tongue covered with fur of this kind very often accompanies chronic gastritis.

(e) A THICK, UNIFORM, MOIST, WHITISH OR YELLOWISH-WHITE COATING with abrupt edges is seen in the early stages of the acute febrile diseases. In consequence of the diminished amount and altered character of the salivary and buccal secretions, this coating presently loses its moisture and becomes dry and darker in color. After a time it separates, leaving the tongue moist and of normal appearance if convalescence has begun, or dry, hard, red or brown, and denuded of epithelium if the fever continues and particularly if the patient falls into the so-called typhoid condition. Under these circumstances the tongue becomes fissured both longitudinally and transversely. In some cases a deep median fissure forms, on each side of which there is a thick, rough, dry, brownish fur, the tip and edges of the tongue being red and denuded; or again the tongue may be dry, red, and glazed. It is protruded upon request tremulously and slowly and, owing to the accompanying mental condition, is not immediately withdrawn. The disappearance of the crusty coating, the redevelopment of epithelium, and the return of moisture are favorable signs. The tongue may be dry, brown, and incrusted in the last stages of chronic diseases of the nervous system, and in cancer, nephritis, and pulmonary tuberculosis.

(f) The thick white fur of the acute febrile diseases is sometimes penetrated by the greatly enlarged filiform papillæ which appear as scattered bright red minute points. This constitutes one of the forms of so-called "strawberry tongue." It occurs with some frequency in scarlet fever, but is not diagnostic of that disease, since it may be present in other acute febrile infections.

(g) A dense, white, flaky coating is sometimes seen upon the tongue of patients who are fed upon an exclusive milk diet. A somewhat similar appearance may be presented by children suffering from thrush—a condition caused by *saccharomyces albicans*, which begins on the tongue in the form of slightly elevated pearly white spots which, by increase in size and coalescence, may cover the greater part of the dorsum of the tongue.

(h) General hypertrophy of the papillæ gives rise to a peculiar appearance which suggests coarse plush. This is the *shaggy tongue*. It is seen in gastro-intestinal and constitutional diseases in advanced life, but is sometimes present in elderly people whose health is good. The shaggy tongue is frequently also fissured, the plush-like surface being divided by conspicuous deep longitudinal and transverse lines of separation. The color is usually deep red. Upon the supervention of acute illness it quickly becomes dry, hard, and full, usually remaining rough.

A red, dry tongue, denuded of epithelium, glistening and resembling raw beef—the *beefy tongue*—occurs in dysentery and chronic intestinal catarrh. It is seen also in hepatic abscess.

Other conditions of the tongue may be of diagnostic importance: fissures, ulcers, mucous patches and plaques, tumors, and cicatrices.

(a) Fissures of the tongue are often seen in healthy persons in advanced life. They may be the signs of a superficial chronic glossitis caused by habitual use of tobacco or irritating food or drink. The median longitudinal fissure is commonly the most marked and readily becomes ulcerated. Transverse fissures are common. Sometimes the fissures are forked or curved. Fissures may be deep and inflamed, the result of extending glossitis—dissecting glossitis—or syphilis. Fissures are common in chronic hepatic disease, chronic colitis, and diabetes mellitus. Local fissures or notches at the edge of the tongue may arise from the irritation of a broken or carious tooth or from syphilitic ulceration.

(b) Ulcers of the Tongue.—Simple excoriations occur as the result of slight traumatism or scalding, or spontaneously in dyspeptic conditions. Aphthous stomatitis is characterized by small, slightly depressed spots with grayish bases and bright red margins. They occur at the edges and tip of the tongue, on the frænum, and elsewhere about the mucous membrane of the lips and mouth. The ulcers are preceded by vesicles and are attended with great pain. The buccal secretions are increased. The ulcers may appear singly or in series or crops. They occur in transient gastric derangements and in women at the menstrual period. There is an individual predisposition to them.

A chronic, recurrent herpetic eruption of the buccal mucous membrane, sometimes associated with erythema multiforme, has been observed in neurotic persons.

Riga's disease is an affection occurring about the time of the first dentition and characterized by a pearly white pseudomembrane beneath the tongue and upon the frænum, with induration and ulceration. It is endemic and sometimes epidemic in Southern Italy.

Superficial ulcers with a red glazed surface occur upon the tongue in various forms of chronic glossitis. They are of irregularly round or oval shape with infiltrated edges and are usually extremely painful. Ulceration of the tongue is commonly attended with salivation. Tuberculosis of the tongue shows itself in the form of circumscribed, indolent, irregularly extending ulceration with a necrotic or caseous base. The edges are usually slightly infiltrated but sharply defined. This ulcer is extremely painful upon contact and is sometimes attended by salivation. The lesions may be single or multiple and are usually secondary to tuberculous disease of the lungs. The glands at the angle of the jaw are not usually enlarged. Syphilis is a common cause of ulceration of the tongue. In secondary syphilis superficial and linear ulcers are common at the border of the tongue as the result of the irritation of the teeth. A single ulcer with an indurated base and enlargement of the cervical glands may be the initial lesion of syphilis. A mucous patch may undergo ulceration, and in later syphilis a gumma may become necrotic, forming a deep foul ulcer. In some instances difficulty attends the differential diagnosis of a single ulcer, which may be due to tuberculosis, syphilis, or malignant disease. The resemblances upon inspection and palpation may be very close. In the first there are usually evidences of tuberculosis of the larynx or lungs and the presence of tubercle bacilli in the scrapings. In cases not otherwise to be determined inoculation experiments should be performed. In the initial lesion of syphilis the induration is dense and circumscribed. The age and habits of the patient are to be taken into consideration. Great enlargement and tenderness of the lymphatics of the neck constitute important symptoms. The evolution of the process and the development of mucous patches, cutaneous rashes, fever, and the like make the diagnosis clear. In gummatous ulceration the enlarged surface is greater and the infiltration less dense. The therapeutic test is important; the ulcer heals under antisyphilitic treatment. A carefully taken clinical history sheds light upon a doubtful case. In epithelioma of the tongue the diagnosis may be reached by exclusion. The process tends to spread, the sublingual lymphatics become involved, the ulcer is foul and indolent, and the patient is almost always past middle age.

The ulcer frequently observed on the frænum of the tongue in whooping-cough is traumatic. It results from the violent impact of the under surface of the tongue against the sharp lower incisors during the paroxysm.

(c) Mucous Patches and Plaques. — The multiple grayish-white superficial lesions of syphilis known as mucous patches occur upon the tongue as well as upon the soft palate, cheeks, and lips. A slightly raised, smooth, red, oval-shaped area sometimes seen in the middle of the dorsum of the tongue in pipe smokers is known as the smoker's patch. The surface is smooth and sometimes white or pearly white in appearance.

Xanthelasma occasionally appears upon the sides of the tongue in the form of yellowish, soft, slightly raised, oblong patches. It occurs in various conditions but is noticeably frequent in chronic jaundice and diabetes.

Leucoplakia is a condition characterized by the development of irregular white or pearly-white smooth patches upon the tongue which show no tendency to ulcerate. They are hard to the touch and gradually extend, sometimes becoming papillomatous. These patches may be the starting-point of epithelioma. The condition is described under various terms, as *buccal psoriasis, ichthyosis* and *keratosis mucosæ oris.* They present some points of similarity to the lesions of syphilitic glossitis, which is, however, more common at the edge and tip of the tongue than on the dorsum and yields to antisyphilitic medication.

Eczema of the Tongue—Geographical Tongue.—This condition is characterized by the formation of irregularly annular patches upon the tongue. There is desquamation of the epithelium. The process is attended with burning and itching. The patches extend at the margins with new formation of epithelium in the centre. The borders are slightly red and well defined but without induration. The condition is more common in infants and children than in adults. The process is recurrent and protracted.

(d) Tumors of the Tongue.—Solid tumors of the tongue are usually tuberculous or syphilitic. They invade the substance of the organ, usually presenting toward its dorsal surface. Tuberculous nodules break down, promptly giving rise to an indolent ulceration with caseation. Gummata rapidly undergo extensive necrosis but yield to treatment. Retention cysts occur in connection with the tongue. Ranula is the most common; it is due to an obstruction and dilatation of a duct of the sublingual or submaxillary glands. Mucous cysts also occur. Echinococcus cysts, which develop as a rule by preference in highly vascular structures, are rare in the tongue. Carcinoma is much more common in men than in women and extremely malignant. Sarcoma is comparatively rare.

(e) Cicatrices. — Scars upon the tongue tell the tale of former traumatism, as the accidental biting of the tongue, a fall or blow upon the chin when the tongue is between the teeth, or the grinding of the teeth during the clonic convulsions of epilepsy. They may be the indications of former active diseases, especially syphilis. Sclerosis of the tongue with local deformity is a common result of the healing of gummatous ulceration.

The buccal mucous membrane is commonly implicated in infections involving the other organs of the mouth, especially the various forms of stomatitis. It is very often the starting-point of the progressive gangrenous affection known as noma or cancrum oris.

VIII.

THE DIGESTIVE SYSTEM (CONTINUED): THE PALATE: TONSILS; PHARYNX.

The passage from the mouth to the œsophagus by way of the pharynx is called the fauces or *isthmus faucium*. It is bounded above by the soft palate, laterally by the palatine arches and tonsils, and below by the base of the tongue. These structures are covered with mucous membrane continuous with that of the mouth and are liable to the same morbid processes. An inspection of these parts yields information of importance in the diagnosis of local and constitutional disease. Infection may take place directly or by extension from the mouth and nasopharynx. Forms of angina —simplex, follicular, suppurative, and diphtheritic—result. When the tonsils are principally or alone involved the condition is spoken of as tonsillitis. The underlying muscular structures may be involved by extension. The tonsils and adjacent lymph structures are points of invasion for the infecting agents in rheumatism and other affections. There are forms of acute tonsillitis that are essentially rheumatic. In children the articular manifestations of rheumatic fever and chorea frequently show a definite relationship to tonsillitis and the latter affection is not rarely followed by endocarditis and chorea. The tonsils may be the port of invasion for tuberculosis or the seat of tuberculous lesions.

Subacute and chronic pharyngeal inflammation may be secondary to gastric disorders or to the gouty diathesis. The pharynx is sometimes involved in rheumatism. Paralysis of the soft palate and spasm and paralysis of the pharynx occur. Superficial ulceration of the pharynx is very common in advanced pulmonary tuberculosis.

General redness of the faucial mucous membrane occurs in simple inflammations and in many of the specific febrile affections, as rötheln, the variolous diseases, influenza, and erysipelas. In the exanthemata, especially measles, scarlatina, varicella, and variola, there are efflorescences corresponding to the cutaneous eruptions. In these situations the pocks of varicella and variola, owing to the action of warmth and moisture, lose their roof in the vesicular stage and are converted into small circular or oval superficial ulcerations with purulent or necrotic bases and a more or less marked areola. Redness of the mucous membrane in this region is a symptom of chronic gastritis or the action of certain drugs, as the iodine compounds and belladonna, and of corrosive poisons.

Hemorrhage occurs into the mucous membrane in the form of petechiæ, infarcts, and extravasations, and there is bleeding from these surfaces in general hemorrhagic states. These tissues are pallid in the anæmias, yellow in jaundice, and show a bluish tint in cyanosis. The mucous patches of syphilis may be seen.

Pain is a prominent symptom in angina, especially in the acute forms. It may be spontaneous, but is excited by the movements of deglutition and by contact of articles of food and drink with ulcerated surfaces. Pain

and tickling referred to the pharynx may be symptomatic of acute rhinitis. These symptoms are common in hay fever. Sensations of dryness and tickling accompanied by the inclination to hawk and clear the throat are constant symptoms of pharyngitis. Annoying hawking is especially excited by disease of the nasopharynx.

Dysphagia is common. It varies in degree and may be due to pain or to mechanical obstruction. When dysphagia is marked both these causes are commonly operative. In suppurative tonsillitis and retropharyngeal abscess dysphagia may be complete. It is a symptom of the various forms of stomatitis and glossitis as well as of tonsillitis and pharyngitis. Painful dysphagia referred to the pharynx is a common symptom in cases showing no signs of inflammation of the mucous membrane—rheumatic pharyngitis. The angina which attends diphtheria, scarlet fever, measles, varicella, and variola is accompanied by dysphagia which is often distressing.

Dyspnœa may become an important symptom in suppurative tonsillitis, retropharyngeal abscess, and erysipelas extending to the pharynx.

Chronic interference with respiration accompanied by mouth-breathing results from hyperplasia of the tonsils and especially from hyperplasia of the pharyngeal tonsil—*adenoid vegetations*. In severe acute angina and in certain chronic diseases involving the tonsils and pharynx, as cancer and forms of syphilis, the drainage of the fauces is interfered with and the accumulating secretions and exudates undergo decomposition. The odor of the breath may be intense, fetid, and disgusting. Accumulations of epithelial cells, leucocytes, and bacteria in the tonsillar crypts are very common in chronic lacunar tonsillitis and in individuals presenting no other symptoms of disease of the throat. They appear as small white or yellowish-white concretions which sometimes undergo calcareous changes. They are sometimes expectorated and should be removed by the curette. They impart a disagreeable odor to the breath.

THE PALATE.

Developmental deformities do not fall within the scope of this work. A narrow, high, arched palate is regarded as among the stigmata of degeneration. Circumscribed ulceration of the mucous membrane of the hard palate is frequently met with in the new-born or may be caused in artificially-fed children by the irritation of the rubber nipple. The ulceration thus caused is sometimes described under the term *Bednar's aphtha*. In young children patches of thrush are not uncommon upon the hard palate. Abscess formation attended with great pain occasionally involves the mucous membrane of the hard palate in connection with alveolar disease. Perforations occur as the result of syphilis.

The soft palate in health is freely movable and symmetrical. The form of the uvula varies in different persons. As a result of defective development it is sometimes bifid. It may be attached laterally to the soft palate or tonsil or to the posterior wall of the pharynx in consequence of adhesive inflammation in diphtheria or syphilis. Perforation of the soft palate is almost always the result of syphilis. In very rare instances it has followed scarlet fever. The uvula varies in length normally. It

frequently becomes elongated in angina and bronchitis. Under these circumstances it causes irritation of the base of the tongue and excites cough, especially in the recumbent posture; the mechanical violence of intense paroxysmal cough elongates the uvula and thus a vicious circuit is established. It becomes elongated and œdematous in cases of debility, anæmia, and anasarca. When greatly œdematous the uvula becomes globular and may attain the size of a cherry, interfering with swallowing and breathing and producing a constant disposition to hawk. In constitutional hemorrhagic states submucous extravasation of blood may occur in the uvula. In very rare instances crops of vesicles resembling herpes show themselves upon the palate.

Anæsthesia of the hard and soft palate and of the anterior two-thirds of the tongue occurs in lesions of the sensory division of the fifth nerve. The tactile sense is usually lost before the pain. The palate is innervated by the accessory nerve to the vagus. Paralysis of the soft palate occurs in bulbar palsy, basal tumors, and meningitis of the base. By far the most common cause is postdiphtheritic neuritis. Upon inspection while the patient pronounces the long *a* the palate and uvula are thrown back and elevated. Under normal circumstances the extent of this movement is the same on both sides. In unilateral paralysis movement upon the affected side is greatly diminished. In bilateral paralysis the whole palate remains relaxed and motionless, the voice has a nasal character, the pronunciation of certain consonants—gutturals—is impaired, and upon attempts to swallow, liquids are returned through the nose. Lesions involving the nerve-supply of one side cause unilateral paralysis.

THE TONSILS.

The tonsils, also called *amygdalæ* from their almond shape, lie at the side of the pharynx between the anterior and posterior palatine folds. They are larger in childhood than in adult life and early undergo senile involution. The greater part of their surface is exposed to inspection by ordinary methods. Upon gagging they are rotated forward. In inflammation the mucosa is reddened and swollen and the surface covered with a mucoid or mucopurulent secretion which may be tinged with blood. In follicular or lacunar tonsillitis this secretion develops in the crypts, producing whitish-yellow spots. These may by extension and coalescence form patches upon the tonsils presenting a superficial resemblance to diphtheria. The pseudomembrane thus formed is not usually distinctly marginate and corresponds in appearance to the points of exudate seen to occupy adjacent crypts. It is not developed in the mucosa but lies upon it, as may be seen upon removing it by wiping or gentle scraping. A pseudomembranous exudate frequently forms in the course of various infections, as scarlet fever, measles, pertussis, enteric fever, and variola. In a great majority of these cases the Streptococcus pyogenes is the active organism. As a rule the development of this form of pseudomembrane does not constitute a serious complication of the primary disease. It may, however, give rise to an intense angina with local sloughing and grave constitutional disturbance. A general streptococcus infection is by no means

infrequent. A pseudomembranous exudate occurs in its most typical form as a manifestation of diphtheria. It is caused by the Klebs-Löffler bacillus.

In suppurative tonsillitis or quinsy one or both tonsils may be involved. The earliest symptoms are those of an ordinary acute angina—pain, dryness, dysphagia, with fever and other symptoms of constitutional disturbance. The tonsils are enlarged, dusky red, and œdematous. They may even meet, or if one only is involved it may extend some distance beyond the median line. In many instances there is salivation. The breath is foul, the glands of the neck enlarged, and the patient opens his mouth only partially and with great difficulty. After suppuration occurs fluctuation may be felt.

Enlargement of the tonsils is common in children. It may be due to repeated attacks of acute tonsillitis or to a chronic inflammatory process leading to a hyperplasia of the lymphoid elements. The tonsillar crypts are enlarged. In some cases a probe may be introduced to the depth of a centimetre or more. Partial or complete adhesions of the anterior pillars to the tonsils are seen, and these structures are sometimes thin, red, and stretched by the enlargement of the tonsil. In some instances the tonsils are dense and firm, the connective-tissue stroma predominating. Enlargement of the tonsils is very often associated with adenoid vegetations in the pharyngeal vault. Mouth-breathing and its concomitant derangements accompany this condition. Ulceration of the tonsils is not very common. In syphilis the primary chancre has occurred upon the tonsils. In secondary syphilis mucous patches are very common in this region, and in the tertiary stage gumma may give rise to enlargement of the tonsil and, upon breaking down, result in deep circular ulceration with a necrotic base and little hyperæmia of the surrounding tissue. Tuberculous ulceration of the tonsils is not common.

THE PHARYNX.

This organ may be divided into an upper portion—the nasopharynx—and a lower portion—the oropharynx. The former may be examined by palpation with the finger or by the rhinoscopic mirror; the latter by direct inspection in a good light. Small foreign bodies, as fish-bones or a beard of wheat, may be recognized upon inspection; larger foreign bodies, as an artificial denture or fragment of meat or bone, by inspection or palpation. The presence of adenoid vegetations due to hyperplasia of the pharyngeal tonsil may be thus determined. Papillomatous masses sometimes fill the vault of the pharynx, extending into the posterior nares and greatly interfering with respiration. By occluding the orifices of the Eustachian tubes they cause deafness and middle-ear disease.

Cyanosis and Pulsation.—Cyanosis of the pharyngeal mucosa may result from general derangements of the circulation or respiration or from local causes, as obstruction to the return flow of the blood by way of the superior vena cava, from aneurism or from mediastinal tumor. In aortic regurgitation pulsation of the capillary vessels may be seen or unilateral pulsation may be the manifestation of a tortuous internal carotid artery or aneurism of that vessel. In the oozing that takes place from the pharynx

in intense congestion or hemorrhagic states the blood may be swallowed and accumulate in the stomach. If vomited the hemorrhage may be attributed to a lesion of the stomach. This error of diagnosis may be avoided by careful inspection of the pharynx.

Pharyngitis.—In acute inflammation of the oropharynx the mucosa is congested and reddened. The patient complains of tickling and dryness with a constant desire to hawk. The secretions are diminished and altered. There is dryness with thin flakes or a whitish exudate, to be seen only upon close examination. The constitutional symptoms are slight.

Rheumatic angina is characterized by sore throat and dysphagia referred to the pharynx. In the majority of the cases the signs upon inspection are not distinctive.

Chronic pharyngitis may develop insidiously or as the result of repeated acute attacks. The mucosa is at first reddened and shows distended vesicles; later it is relaxed and presents a granular or warty appearance—granular pharyngitis, due to hyperplasia of the lymph elements. The secretion is mucoid or purulent and undergoes desiccation, forming dry crusts or scales which very often communicate an offensive odor to the breath. The process extends into the nasopharynx. There is very often a free mucoid or mucopurulent secretion which gives rise to the sensation of dropping or trickling into the throat and causes hawking. In other cases the secretion is slight and the mucous membrane reddish-brown, dry, atrophic, smooth and glistening—*pharyngitis sicca*. The pseudomembranous exudate of diphtheria frequently extends into the pharynx; the exudate of pseudodiphtheritic, diphtheroid, or diplococcus inflammation commonly appears upon the tonsils and does not as a rule involve the pharynx.

Ulceration of the pharyngeal wall is not uncommon. Limited areas of superficial ulceration occur in chronic pharyngitis. Small round or oval ulcers upon the posterior wall are sometimes seen in enteric fever. Irregular superficial patches of ulceration are frequently seen in the later stages of consumption. The bases are necrotic and grayish-yellow. The ulceration may involve the greater part of the posterior pharyngeal wall and cause intensely painful dysphagia. Ulceration of the pharyngeal wall occurs also in syphilis. In the secondary stage it is very often superficial and associated with mucous patches. In the tertiary stage it results from the breaking down of gummata which heal satisfactorily under treatment, leaving white cicatrices.

Ulceration of the pharynx may occur in connection with the various forms of pseudomembranous inflammation and attends cancer and lupus. The etiological diagnosis of ulceration of the pharynx is frequently attended with difficulty. As in the case of the tongue, tubercle, cancer, and syphilis are to be differentiated. A careful anamnesis is important. The associated clinical phenomena are very often characteristic. In tuberculosis the presence or absence of tubercle bacilli and the inoculation test are important; in syphilis the therapeutic test.

Acute phlegmonous inflammation of the pharynx may result from traumatism or foreign bodies in the pharynx.

Acute infectious phlegmon, a rare condition, characterized by anginal symptoms, dysphagia, rapid abscess formation, swelling of the neck, and

severe constitutional symptoms, may result from direct traumatism, the injury caused by foreign bodies, or arise spontaneously.

Retropharyngeal abscess manifests itself upon inspection and palpation as a projecting fluctuating tumor upon the posterior wall of the pharynx in the median line. Attendant phenomena are restlessness, dysphagia, and changes in the voice, which becomes nasal or metallic as the result of pressure. Retropharyngeal abscess is a rare affection. It has been observed in children previously in apparent good health as a sequel of the infectious diseases, particularly scarlet fever and diphtheria, and in caries of the cervical vertebræ.

Angina Ludovici: Ludwig's Angina; Cellulitis of the Neck.—A rapidly developing phlegmonous inflammation of the tissues about the floor of the mouth is described under these names. It apparently results from trauma or some lesion about the roots of the teeth or from infection of the submaxillary gland. It may occur as the result of secondary infection in the specific fevers, particularly diphtheria and scarlet fever. The inflammation is the result of streptococcus infection. Swelling usually appears first in the submaxillary region of one side and rapidly spreads, with diffuse dull redness and brawny induration of the neck. The tendency is to speedy suppuration and extensive gangrene with general septicæmia. The disease is rare and very fatal.

The innervation of the pharynx is derived from the pharyngeal plexus, formed by the combination of the glossopharyngeal and branches of the vagus.

Spasm of the pharynx is a functional disorder. It is common in neuropathic individuals. It is the cause of ordinary gagging and occurs in hydrophobia and as a convulsive manifestation of hysteria—*globus hystericus*.

Motor palsy of the pharynx occurs in postdiphtheritic neuritis, acute ascending paralysis, and bulbar paralysis. It may result from lesions at the base of the brain. It is commonly bilateral. There is difficulty in swallowing and food is not properly passed into the œsophagus. Particles of food may pass into the larynx and, when there is associated paralysis of the soft palate, into the posterior nares. Fluids are regurgitated through the nose. In unilateral lesions the power of deglutition remains.

Anæsthesia of the pharynx is produced by bromidism and the local application of cocaine.

IX.

THE DIGESTIVE SYSTEM (CONTINUED): THE ŒSOPHAGUS.

The upper limit of this organ is about at the level of the cricoid cartilage and opposite the sixth cervical vertebra. It terminates in the cardiac orifice of the stomach opposite the upper border of the body of the eleventh thoracic vertebra. It has a short infradiaphragmatic course of about one and a half centimetres. It begins about six inches—fifteen cm.—from the incisor teeth, is about nine and a half inches—twenty-four cm.—in length, and varies from three-fourths to one and a fourth inch—two to three cm.—in diameter, the narrowest parts being at the commence-

ment, in the middle, where it is crossed by the left primary bronchus, and at its point of entrance into the stomach. The œsophagus is in relation with the trachea, the left bronchus, the thyroid body, the peribronchial lymph-glands, the pneumogastric and recurrent laryngeal nerves, the aorta, the azygos vein, the thoracic duct, and the pericardium and pleuræ. Nearly its whole course is in the posterior mediastinum.

The principal methods of examination are auscultation, direct inspection of the interior of the œsophagus, the use of the sound, and the Röntgen rays. Ordinary inspection, palpation, and percussion are of no practical value, although the first two of these methods may reveal a tumor upon the left side of the neck when there is a diverticulum or new growth in the cervical portion.

Auscultation. — Upon auscultation, the stethoscope being placed to the left of the ensiform cartilage or to the left of the spine opposite the tenth rib, a gurgling sound may be heard six seconds after the act of swallowing, as determined by the movement of the larynx. This murmur is due to the propulsion of the liquid or bolus of food into the stomach and is not to be confounded with the sound to be heard over the cervical part of the œsophagus during swallowing. The absence, delay, or prolongation of the first-named sound is evidence of obstruction at the lower end of the œsophagus.

Œsophagoscopy. — Direct inspection may be practised through a suitable tube or instrument with proper illumination. The mucosa in acute inflammation is reddened, swollen, and lax; in chronic inflammation, grayish-white, covered with a viscid mucus, and shows dilated veins. The instrument may be used as a sound to determine the presence or absence of dilatation or narrowing. Ulceration, new growths, and cicatrices may be recognized, and fragments of ulcerated tissue have been removed through the œsophagoscope for examination. Foreign bodies may be located and have been removed by instruments passed through the tube when their removal by ordinary methods has proved impracticable.

The Œsophageal Sound. — The ordinary rubber tube used in the examination and treatment of diseases of the stomach may be utilized or œsophageal bougies especially made for the purpose. The latter are of whalebone or narrow blades of metal with rounded edges and provided with adjustable olive-shaped tips made of hard rubber, ivory, or metal and of various sizes. The sound is introduced in the same manner as the stomach tube. It may pass directly into the stomach or be arrested by some obstruction. The location of the stenosis can be readily determined by measuring the distance from the teeth upon withdrawing the instrument. No force is to be used. Feeble and anæmic patients may faint during this examination and neurotic or hysterical individuals may have local spasm or even general convulsions. Under such circumstances the instrument should be immediately withdrawn. Sounding must be performed with due caution, since there is the danger of injury or perforation of the wall of the œsophagus, the rupture of an aneurism, or the laceration of the varicose veins of the œsophageal plexus in atrophic cirrhosis. By the use of the sound the location of strictures, dilatations, diverticula, ulceration or at least areas of sensi-

tiveness, and the presence or absence of foreign bodies and their location may be learned. The careful use of this instrument yields information as to whether or not a stricture is dilatable or rigid and unyielding.

The X-rays.—The presence and position of foreign bodies in the œsophagus may be ascertained by this method of examination, and in appropriate cases information in regard to tumors of, or in relation with, the œsophagus. The possibility that a large atheromatous plate in the aorta may be mistaken for a foreign body in the gullet is to be borne in mind.

Symptoms of disease of the œsophagus are dysphagia, pain, and the regurgitation of food.

Dysphagia varies according to the disease and its site and is commonly greater with solids than with fluids; the pain may be sharply localized or diffuse; the regurgitation of food may be partial or complete and take place immediately or not for some time.

The œsophagus is subject to developmental defects, of which the most important is atresia. Liquids are immediately regurgitated and the sound cannot be passed. Death results from inhalation pneumonia or starvation.

Alterations in Calibre.—The œsophagus may be narrowed or dilated. Very often these two conditions are combined, the tube being narrowed at one point and dilated at another.

Narrowing may be intrinsic, due to lesions of the œsophagus itself, as congenital defect, stricture from inflammation, cicatrix or neoplasm, or muscular spasm; or extrinsic, due to pressure from without. The symptoms vary. In the first instance they are chiefly dysphagia, pain, and regurgitation; in the second there are superadded to these the symptoms of the disease causing the compression. Narrowing may be a congenital defect. Its position in this case is usually at the upper or lower extremity. The chief symptom is dysphagia.

In **inflammatory and cicatricial stenosis** there is a history of accidental or intentional swallowing of a caustic or corrosive fluid, or the history may point to ulceration as the result of traumatism produced by a foreign body, softened glands, syphilis, or peptic ulcer at the cardia. Ninety per cent. of the cases of stenosis are due to cancer, which acts by infiltrating the walls and causing the development of contracting connective tissue. In stricture arising from cicatrix the dysphagia comes on gradually and is progressive and permanent. It may begin abruptly and at first be caused by solids only; later by fluids. Associated spasm may cause variations in degree, but there are no intervals of complete relief as in spasmodic stricture. The bougie is always arrested at the same distance from the teeth. Food is regurgitated shortly after it is taken and, unless acid in itself, shows an alkaline reaction. Subjectively it seems to stop at or near the manubrium. There is actual progressive starvation and corresponding emaciation. Signs of pressure upon the recurrent pharyngeal nerves are rare in cicatricial stenosis.

Malignant stricture of the œsophagus is commonly carcinomatous. A limited number of cases of sarcoma have been reported. Carcinoma is more common in men than in women. It is rare before forty and most

frequent between fifty and sixty. It occurs with about equal frequency in the upper and the lower half of the organ. The symptoms are not very different from those of cicatricial stricture. Pain is more prominent; it is usually referred to the gullet; sometimes to the back between the shoulder-blades. The food is commonly returned shortly after it is taken and is sometimes streaked with blood, or it may contain fragments of necrotic tissue. The obstruction may become complete by the impaction of food in the stricture. Cough is common, and hoarseness, aphonia or complete loss of voice may result from involvement of the recurrent laryngeal nerves. Hunger, at first urgent, gives place to indifference to food. Thirst is troublesome, the mouth dry, the breath foul, and hiccough frequent. The progress of the disease is rapid.

Spasmodic Stricture.—Œsophagismus occurs in neurotic persons and especially in hysterical women. It has some points of resemblance to the "globus hystericus." It may be due to mental shock or prolonged depressing emotions, but it is more frequently due to reflex irritation in disorders of the gastro-intestinal or reproductive tract. In rare instances it accompanies disease of the larynx, and it often recurs in diseases of the œsophagus. It occurs in human rabies and in the hysterical counterfeits of that disease, and has been observed in cerebrospinal fever, tetanus, and epilepsy. Dysphagia is paroxysmal and of varying degree. It comes on abruptly and often passes away as rapidly as it came. Food is regurgitated suddenly and with force. There are gulping sounds. The difficulty in swallowing is produced by liquids as well as by solids. The patients often complain of pain which is constricting and burning in character. Emaciation does not usually occur. The bougie is not always arrested at the same level and may usually be passed by firm pressure beyond the point of resistance and into the stomach.

Pressure.—Narrowing of the œsophagus by pressure from without may be caused by enlargement of the thyroid body, as in goitre, Graves's disease, cystic degeneration, or tumors involving that organ or enlarged lymph-glands. Within the thorax a mediastinal tumor, dislocation backward of the sternal end of the clavicle, prevertebral abscesses and tumors, aneurism of the aorta, a distended diverticulum or massive pericardial effusion may compress the œsophagus. The essential symptom is dysphagia. The sound may usually be passed with persistent gentle pressure. If there is reason to suspect the presence of an aneurism the sound must not be used. The œsophagus adjusts itself to external pressure to a remarkable degree and unless it is extreme the symptoms are slight.

Obstruction from plugging may result in infants from excessive proliferation of the thrush fungus; at any period of life from any foreign body swallowed by accident or design. Common among these are masses of meat, fragments of bone, artificial dentures, jack-stones and other small playthings. Pedunculate polypi and other tumors may obstruct the œsophagus without causing stricture.

Diverticula or circumscribed lateral dilatations are of two kinds, those caused by internal pressure—*pulsion diverticula*—and those brought about by the contraction of fibrous tissue outside the organ—*traction diverticula.*

Pulsion diverticula first show themselves by discomfort or a sense of obstruction after swallowing food, usually referred to the sternal region and often attended by cough. After a time liquids only can be taken and are sometimes regurgitated and swallowed again and again before reaching the stomach. Portions of food may be regurgitated several hours after having been swallowed. Pressure or upward stroking of the left side of the neck may aid in the regurgitation of food. A tumor is not often present. Upon auscultation the sound produced by the passage of food into the stomach is absent. A sound may be introduced into the diverticulum, the blind end of which may be eight inches—twenty cm.—or more from the teeth; it may pass into the stomach and be freely movable in that organ; or one sound may be passed into the diverticulum and while it is still in place another may be passed beyond it into the stomach. The symptoms increase in severity and in many cases there is progressive emaciation. As a rule the progress of the disease is tardy.

Traction diverticula are usually situated upon the anterior or lateral wall and near the bifurcation of the trachea. They are funnel-shaped and vary in depth from one-half to three-quarters of an inch and are usually single but may be multiple. They commonly give rise to no symptoms. Particles of food or foreign bodies may, however, be caught in them and cause ulceration and perforation, with bronchopneumonia, pulmonary gangrene, mediastinitis or pericarditis and pleurisy. A positive diagnosis cannot be made.

Ulceration may cause tenderness, dysphagia; perforation the secondary lesions just mentioned; and rupture, which usually results from the presence of a foreign body, may cause gangrenous mediastinitis and pleurisy.

Œsophageal hemorrhage may result from ulcer, cancer, the presence of foreign bodies, the rupture of an aneurism or of the dilated veins of the œsophageal plexus in thrombosis of the portal vein or in atrophic cirrhosis of the liver.

The bleeding may be occasional or constant and vary in quantity from a trifling amount to a copious loss which is quickly fatal. The associated symptoms may render the diagnosis easy, but in the case of varicose veins the differential diagnosis from gastric hemorrhage is often difficult. The presence of other symptoms of portal obstruction and the fact that the blood is regurgitated rather than vomited are to be considered, but the blood may be discharged into the stomach and subsequently vomited.

Inflammation of the Œsophagus.—Œsophagitis is attended by localized or diffuse pain upon swallowing, prostration, and in the severe acute forms by chills and fever. There may be tenderness upon pressure and upon bending the spine; for this reason the head is held rigid. Abscess formation may show itself by a circumscribed swelling upon one side of the neck with pressure upon the larynx and hoarseness and dyspnœa. In the phlegmonous form pus may be expectorated and in the chronic cases a glairy, viscid mucus.

Tuberculous and syphilitic ulcerations occur as local manifestations in these diseases, and their presence is to be suspected when there is dysphagia, persistent substernal pain, or the regurgitation of blood-stained mucus in connection with the general phenomena of these diseases respectively.

Paralysis. — The œsophageal muscles are sometimes paralyzed in central or peripheral diseases of the nervous system. Lesions in the neighborhood of the origin of the pneumogastric nerves, such as hemorrhage, softening, tumor, or sclerosis, are among the central causes; pressure neuritis of the pneumogastric from enlargement of the lymphatic glands, or disease of the vertebræ and toxic neuritis after diphtheria or in chronic alcoholism or lead poisoning are among the peripheral causes. Difficulty of swallowing, without pain, is a characteristic symptom. It develops gradually or abruptly, according to the cause. The food produces a sense of weight or pressure and a large bolus is more readily swallowed than small morsels. Fluids may be regurgitated. Gurgling sounds attend the act of swallowing, but the normal sound at the cardia is not heard. The bougie passes freely.

X.

THE DIGESTIVE SYSTEM (CONTINUED): APPETITE; THIRST; ERUCTATIONS; REGURGITATION; NAUSEA; VOMITING; THE VOMITUS; DEFECATION; CONSTIPATION; DIARRHŒA; TENESMUS; PAINFUL DEFECATION; FECAL INCONTINENCE; CHARACTER OF THE DISCHARGES.

Appetite, thirst, the frequency of defecation and the consistency and other characters of the stools vary within wide limits in health. They are to a considerable extent influenced by habit and the mode of life of the individual. Beyond these limits they, together with certain associated symptoms, acquire clinical significance of importance in disease of the digestive organs and other local and general affections.

APPETITE.

Appetite is dependent upon the state of the gustatory nerves, the condition of the stomach, and the requirements of the organism as a whole.

The appetite for food may be diminished, lost—*anorexia;* increased—*polyphagia* or *bulimïa;* perverted—*pica* or *parorexia*; or insatiable—*acoria.*

Loss of appetite varies from mere indifference to food to complete anorexia. It is symptomatic of the most varied morbid states, the enumeration of which would comprise a nosological system.

The appetite is more or less impaired in:

(a) The acute infections. The absence of the normal desire for food is due chiefly to the toxæmia, and in part to the subacute gastritis which is usually present.

(b) The chronic infections, especially in the active stages of syphilis, tuberculosis, and malaria, and in the conditions of malnutrition and cachexia to which these diseases give rise.

(c) Septic conditions, both acute and chronic, and in all forms of local suppuration. Loss of appetite in the absence of gastric disease or

other adequate obvious cause, especially when associated with persistent leucocytosis, may be symptomatic of local suppuration in some part of the body.

(d) Pyrexia. Fever is attended by loss of appetite as in any of the foregoing conditions, and this symptom is commonly present in the early convalescence from febrile diseases. A notable exception to the latter statement occurs in enteric fever, in which hunger is usually a prominent and urgent symptom after the defervescence.

(e) All forms of anæmia, chronic wasting diseases, and in many functional and organic diseases of the nervous system. The loss of appetite is not only an important symptom in these conditions but it is also an etiological factor. A vicious circuit is established. The inability to take food aggravates the condition that causes it.

A remarkable suppression of the desire for food is manifest in certain cases of hysteria. There are instances in which the suppression of appetite is maintained for long periods, as in "fasting girls." Deception is to be guarded against. To hysteria is to be referred the condition described by Gull as *anorexia nervosa*, in which there is not only complete loss of appetite but also absolute inability to take food, with the gravest symptoms of inanition, sometimes ending in death.

(f) Cachexias and terminal states. The patient is not only unable to take food save in minimum quantities, but life is also often maintained without it for considerable periods—a fact due to the extreme limitation of vital activities.

(g) Toxic conditions. Complete loss of appetite attends all acute toxic conditions, and there is great impairment in chronic intoxications, as that of lead, arsenic, or mercury. The anorexia is due in part to the general malnutrition, in part to local disorder of the organs of digestion. In chronic alcoholism appetite is irregular and enfeebled and at the close of a debauch is completely lost. Aversion to food is frequently the forerunner of an attack of delirium tremens.

(h) Psychic states. Depressing emotional conditions, such as result from worry, anxiety, suspense, and grief, are usually attended with anorexia. The impairment of appetite under these circumstances is largely dependent upon the temperament of the individual.

(i) Functional or organic disease of the stomach. Appetite may persist normally or in some abnormal form in the gastric neuroses, and is maintained in some cases of gastric ulcer. Patients suffering from disease of the stomach frequently have a craving for food which is immediately dispelled upon attempts to eat. Loss of appetite not infrequently results from a monotonous or inadequate dietary. Under these circumstances the appetite frequently returns when the patient is permitted to take ordinary food. There is a French proverb to the effect that *appetite comes with eating*.

Polyphagia is a term used to indicate excessive or voracious eating. It may be occasional, as in the convalescence from enteric fever or in children suffering from whooping-cough, the frequent vomiting caused by the paroxysms not permitting the absorption of sufficient food to meet the needs of the organism; or persistent, as in diabetes.

Bulimia and acoria are terms used to designate an insatiable appetite. This is symptomatic of certain insanities and some forms of idiocy and occurs in paroxysms in certain cases of hysteria, neurasthenia, epilepsy, and exophthalmic goitre. In polyphagia the patient eats large quantities of food and is for the time being satisfied. In bulimia the ordinary sense of satiety after eating does not occur. Acoria is the loss of the sensation of satiety.

Pica or parorexia is a craving for unnatural articles of food—a depraved appetite. It is seen in some cases of hysteria, chlorosis, and in pregnancy. These terms are also employed to designate a nervous craving for special articles of diet or for articles that are not fit for food.

THIRST.

Thirst is in some instances an individual peculiarity. There are persons who rarely experience the sensation of thirst and do not consume enough fluid to fully meet the requirements of the body; others who without impairment of health manifest an habitually abnormal desire for fluid.

Impaired Thirst.—The sensation of thirst is diminished in soporous states, even when the buccal and salivary secretions are diminished and the mouth and tongue are dry, as in enteric fever.

Increased thirst is symptomatic of many morbid states. It is a constant symptom in fevers and occurs in all conditions attended with abundant or profuse loss of fluids, and is proportionate to the dehydration of the tissues. It therefore attends profuse sweating both physiological and pathological, abundant diuresis from any cause, persistent vomiting, abundant watery discharges from the bowels, and sudden copious hemorrhage. It occurs at the time of crisis from acute diseases, as croupous pneumonia; in the polyuria of hysteria and persistently in diabetes insipidus and mellitus; in the copious vomiting of acute irritant poisoning and in some cases of uræmia; after the action of elaterium and other drugs producing large watery discharges from the bowels; in cholera nostras and Asiatica and after all kinds of abundant hemorrhages both pathological and traumatic. An unusual desire for water is observed in some cases of chronic gastritis. Persistent excessive thirst is very often the first symptom to attract attention in diabetes. The arrest of the buccal secretions in xerostomia or dry mouth gives rise to continued and distressing thirst. Polydipsia is a term used to describe the habitual taking of fluid in excessive amounts.

ERUCTATIONS, REGURGITATION, NAUSEA, AND VOMITING.

The œsophagus enters the stomach at an angle, forming a valve-like fold which serves to prevent the return of the contents of the stomach. The relation of the central tendon of the diaphragm to the œsophagus is such that it closes the œsophageal opening only at the time of inspiration.

Eructations or Belching.

The spasmodic forcible discharge of gas or air from the mouth is a common symptom. It may come from the œsophagus; much more commonly it comes from the stomach. It is sometimes odorless, frequently offensive. It may consist of air swallowed with the food or with the saliva, or of the gaseous products of the chemical decomposition of the food in the stomach. The eructations may be occasional or occur in paroxysms lasting for periods of some hours. Eructations are symptomatic of acute indigestion such as results from over-eating, various forms of gastritis and other organic affections of the stomach, or they may be of nervous origin.. Large quantities of odorless gas are sometimes forcibly expelled at intervals during a period of several hours in hysterical and neurasthenic individuals, the stomach at the same time being tensely distended.

Regurgitation.

The liquid portions of the food, and in some instances the solids, are returned to the mouth without the violence characteristic of vomiting. Regurgitation from the œsophagus occurs as a symptom of stricture, dilatation or diverticulum, the food being returned immediately or after an interval. Regurgitation from the stomach may be due to over-distention with food, or drink and relaxation of the cardiac orifice. The regurgitation of considerable quantities of an opalescent, slightly alkaline fluid is spoken of as *water-brash.*

Merycism or rumination is the regurgitation of solid food from the stomach to the mouth, when it is again chewed and swallowed. The food is returned in small portions without nausea. This phenomenon appears at first to be the result of regurgitation, later a habit.

Pyrosis or heart-burn is a burning sensation behind the sternum, extending to the pharynx. It is often accompanied by eructation and sometimes by the regurgitation of an acid fluid. It is due to the ejection of the gastric contents into the œsophagus. It occurs in hyperchlorhydria but may appear as a neurosis when the gastric secretion is normal.

Nausea.

Nausea or sickness at the stomach is closely associated with vomiting in its mechanism and clinical significance. It belongs to the group of abnormal sensations referred to the stomach, and occurs in functional and organic affections. Those causes which excite vomiting also excite nausea, though the latter may occur in the absence of the former. The term "nervous nausea" is applied to this symptom when it arises in constitutional disorders and diseases of the central nervous system. It is common in neurasthenia and hysteria and is very often the result of reflex irritation in distant organs, for example, the uterus and ovaries. Nausea is very common in the early stages of pregnancy, and, associated with retching and vomiting, constitutes in pregnancy the syndrome known as *morning sickness*, which in exceptional cases is persistent and intractable and may even cause death.

Vomiting.

Vomiting is the forcible expulsion of the contents of the stomach through the mouth. In exceptional cases the contents of the intestines may also be expelled through the mouth—fecal or stercoraceous vomiting.

The Muscular Mechanism.—The act of vomiting is a complex reflex movement in which many muscles take part. There is usually a sensation of nausea and a reflex flow of saliva into the mouth, accompanied or followed by a series of more or less violent retching movements which consist of deep inspirations with closure of the glottis. As a result of these movements the stomach is compressed by the diaphragm and the negative pressure in the thorax and especially in the œsophagus is decidedly increased. In the course of these retching movements the act of vomiting is brought about by a sudden convulsive contraction of the abdominal muscles which exerts additional pressure upon the stomach. With this the cardiac orifice of the stomach is dilated and the stomach contents are forced through the œsophagus, the glottis being closed by the adductor muscles and the nasal chambers shut off from the pharynx by the contraction of the posterior pillars of the fauces upon the palate and uvula. In the vomiting of unconsciousness, as in anæsthesia, the laryngeal muscles may relax and vomited matters be insufflated into the trachea, and in violent vomiting the material may in part be forced past the palate and uvula and ejected through the nose.

It is not uncommon for the contents of the duodenum to be forced by the violence of the contraction of the abdominal muscles through the pylorus, so that the vomitus consists of bile-stained material and sometimes of pure bile.

The muscles concerned in vomiting are respiratory. The act consists essentially in the simultaneous spasmodic contraction of the diaphragm, an inspiratory muscle, and the abdominal or expiratory muscles, contraction of the muscular fibres of the stomach being altogether of subsidiary importance.

The Nervous Mechanism.—The reflex nature of vomiting is shown by the frequency with which it is produced by the stimulation of sensory nerves and by injuries to various parts of the central nervous system. Disagreeable emotions and derangements of the equilibrium of the body, irritation of the mucous membrane of various parts of the alimentary canal, pathological states of the genito-urinary tract, and lesions or injuries of the brain may all cause vomiting. Vomiting may also be caused by direct action upon the medullary centres, as in the case of drugs—apomorphine and various narcotics—and by or in the toxæmia of the infections and autointoxications, as uræmia and cholæmia.

The causes are many, but the most common is irritation of the sensory fibres of the gastric mucous membrane. In this case the afferent path is by way of the sensory fibres of the vagus; the efferent path by way of the motor fibres innervating the muscles concerned in the act of vomiting, namely, the vagi, the phrenics, and the spinal nerves distributed to the abdominal muscles. It is now generally conceded that there is a definite vomiting centre situated in the medulla in close proximity to the respiratory centre.

The readiness with which children vomit is due in part to the greater reflex excitability of the nervous system in early life; in part to the position of the stomach, which is more nearly vertical than in adults. The undeveloped state of the fundus and the defective closure of the cardia increase the liability of infants to vomiting, which often occurs without effort as a mere regurgitation of a portion of the food upon change of posture or slight pressure upon the epigastrium.

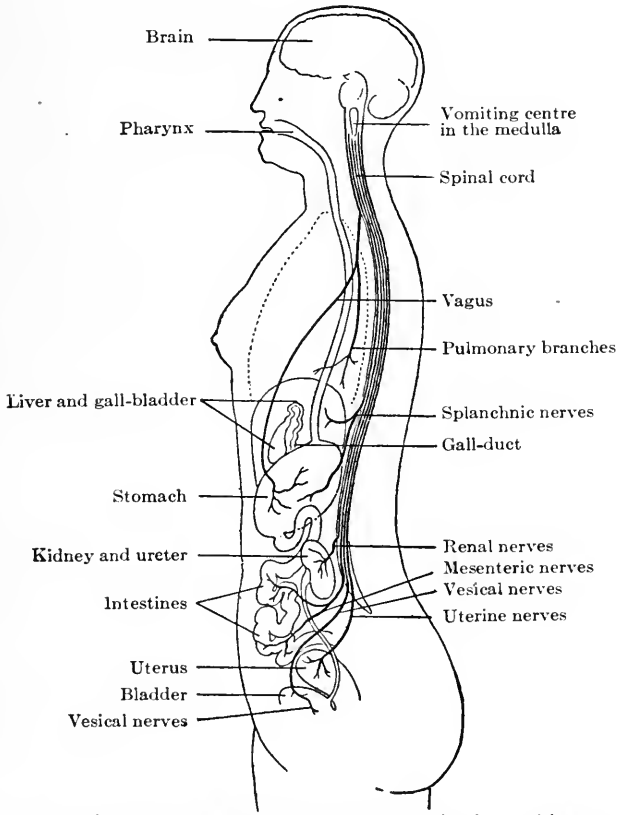


Fig. 200.—Diagram of afferent nerves which may excite the vomiting centre. Modified from Brunton.

1. **Vomiting from Direct Irritation of the Terminal Fibres of the Vagus in the Stomach.**—Vomiting from this cause is very common. It may result from anatomical lesions of the stomach itself and from quantitative and qualitative abnormalities of the contents of the organ. Vomiting is a common phenomenon in various forms of gastritis. In acute gastric catarrh there is vomiting of the gastric contents followed by mucus often stained with bile; a sense of relief is then experienced. In chronic gastric catarrh vomiting is common; it occurs at various intervals after the taking of food. Frequently, and especially in the gastric catarrh of alcoholic subjects, there is distressing vomiting of tough mucus on rising—*vomitus matutinus potatorum*. Vomiting is common in peptic ulcer of the

stomach and is frequently provoked by the intake of food, which also causes pain. The pain very often precedes the vomiting and is relieved by it. The vomiting which attends carcinoma ventriculi is a common and distressing symptom. It is not often present until the disease has made considerable progress. It may occur when the stomach is empty, but usually follows the ingestion of food, after varying intervals. When the growth involves the cardia food may be immediately vomited; when the pylorus, after an interval of several hours. Vomiting may be absent in carcinoma of the fundus or lesser curvature. In stenosis of the pylorus from carcinoma or other cause food is retained in the stomach, which gradually undergoes dilatation, and is vomited after some hours or a day or two—*retention vomiting.* The vomiting of large quantities of fluid after considerable intervals of time is characteristic of gastric dilatation. Vomiting does not occur in gastrectasis of slight degree and in the extreme cases, by reason of the impaired contractility of the wall of the stomach, may wholly cease—an unfavorable symptom. Vomiting is a common symptom of cholera morbus and cholera Asiatica and may be regarded as the direct result of the inflammation of the gastric mucous membrane. It usually occurs after the diarrhœa, sometimes coincidently with it, scarcely ever before it. Vomiting in cholera is usually unattended with effort, is frequently repeated, and ceases or alternates with singultus in the algid stage. The vomitus is liquid and sometimes resembles the rice-water discharges. This symptom occurs in hyperacidity and hypersecretion and may be so persistent in cases of gastric hyperæsthesia that all food is promptly ejected. External pressure upon the stomach, as in pericarditis, ascites, or pericardial effusion, may cause vomiting.

2. **Vomiting from Central Irritation of the Vagus.**—To this cause must be referred the vomiting which is so common in diseases of the brain and its membranes—anæmia, hyperæmia, concussion, sea-sickness, Ménière's disease, tumor, abscess, and various forms of meningitis. So-called cerebral vomiting is characterized by the absence of nausea, its suddenness, projectile character, and the fact that it occurs independently of the taking of food. Vomiting of gastric origin is mostly followed by a sense of relief, while that dependent upon cerebral causes usually aggravates the symptoms, probably because of the mechanical disturbance produced by the act. Vomiting is an early and important symptom in tuberculous meningitis and cerebrospinal fever.

3. **Reflex Vomiting.**—The following forms are to be considered:

(a) Vomiting produced by irritation—tickling—of the base of the tongue or the fauces. Nausea, gagging, and vomiting are frequently caused by the unskilful use of the tongue-depressor or the laryngoscopic mirror. In the older medicine tickling the throat with a feather often played the part of an emetic. When the mucous membrane is abnormally sensitive, as in neurotic individuals or as the result of acute or chronic catarrh, very slight irritation of the fauces may cause vomiting. The vomiting of acute angina, that caused by efforts to dislodge tough masses of mucus, that attendant upon hypertrophy of the tonsils, and the vomiting which accompanies the paroxysm of pertussis must be referred to this group. The irritation caused by partially detached diphtheritic membrane sometimes

produces efforts at vomiting which may have the favorable effect of wholly detaching the mass.

The vomiting of consumption is sometimes an early symptom; it is more common and troublesome in the later stages. It is frequently caused by severe paroxysms of coughing. The vomiting of phthisis may be cerebral, as from tuberculous meningitis, of which it is often an early and ominous symptom; due to pressure upon the vagi by caseous glands; the manifestation of irritation of the peripheral distribution of the vagi; pulmonary, pharyngeal, or gastric or mechanical, as from the succussion of urgent cough.

(b) The vomiting of peritonitis, which is frequently severe and intractable and always significant.

(c) That caused by irritation of the intestinal mucous membrane. In some instances the action of purgatives is preceded by vomiting. This symptom may attend intestinal parasites, colic, enterocolitis, appendicitis, strangulated hernia, intussusception, torsion, and ileus. In any form of obstruction of the bowel retroperistalsis may occur with vomiting, which gradually becomes stercoraceous.

(d) That attendant upon visceral diseases of various kinds, as biliary and renal colic, acute nephritis, pyelitis, cystitis, Addison's disease, and acute yellow atrophy of the liver.

(e) That which is symptomatic of disorders of the female sexual organs. Vomiting is common in anomalies of menstruation, uterine displacements, and pelvic exudates and new growths.

Of especial importance is the vomiting of pregnancy. A little mucus is thrown up with great nausea and effort when the patient rises in the morning. Commonly the vomiting does not recur until the next day; sometimes it is persistent and distressing. Usually it ceases after a few months. The pernicious form has been spoken of above.

(f) So-called nervous vomiting. The most typical form is that which occurs in hysteria. It depends upon the hyperæsthesia and abnormal motility of the stomach and upon quantitative and qualitative changes in the gastric secretions. The vomitus is often of large amount and consists of thin fluid. It is a notable fact that notwithstanding persistent vomiting hysterical patients lose little weight.

The persistent vomiting of Leyden is a form of nervous vomiting characterized by recurrent attacks coming on without obvious cause or as the result of slight indigestion, fatigue, or worry, and lasting from some hours to several days. The vomiting is copious and continuous; the abdomen retracted and the bowels constipated. There is epigastric pain together with intense headache and intolerance of light and sound. The pulse is frequent but there is no fever.

The gastric crises which occur in tabes, and less frequently in acute myelitis, disseminated sclerosis and paresis, are to be mentioned in this connection. Together with distressing pain there is vomiting, usually persistent and uncontrollable. Food is at first ejected, then a colorless stringy fluid and in some cases a blood-stained mucus. There is vertigo and a sense of sinking at the pit of the stomach. The attack lasts from some hours to two or three days. In the intervals there may be no signs of gastric disease.

The vomiting of migraine belongs to the category of nervous vomiting.

(g) Reflex vomiting may accompany diseases of the heart, especially myocarditis, fatty heart and angina pectoris. Vomiting due to cardiac disease is not infrequently associated with hiccough.

4. **Direct Irritation of the Centre for Vomiting.** — This form is less frequent. It arises under the following conditions:

(a) The action of certain emetics of which apomorphine is a type.

(b) The action of toxic substances in the blood, as for example those present in nephritis both acute and chronic. Vomiting is an early and ominous symptom in many cases of uræmia and not rarely the first indication of contracted kidneys. Uræmic vomiting occurs independently of the taking of food and is often severe and distressing.

(c) As an early manifestation of the toxæmia of the acute infections, especially in childhood. Vomiting may attend the stage of onset in scarlet fever, croupous pneumonia, diphtheria, and other acute febrile diseases.

The Gross Characteristics of the Vomitus.

The general appearance, quantity, odor, and reaction of the ejected material is of importance in diagnosis. These peculiarities depend largely upon the presence or absence of food in the stomach, its character and the time that has elapsed since its ingestion. When vomiting occurs directly after eating, the food shows little or no change. On the other hand, if some hours have elapsed there may be no trace of food. In retention vomiting, however, it is not uncommon to find particles of food taken at a previous meal or upon a preceding day. In sucklings the appearance of the vomited milk is of importance. The presence of curds indicates the presence of the milk-curdling ferment; an uncurdled milk some time after nursing shows the absence of normal gastric secretions and may be the sign of grave changes in the stomach.

Aside from the presence of food the following peculiarities are of diagnostic importance:

Watery Fluid and Mucus.—The vomitus may consist of a watery fluid containing little or no mucus. This is common in the morning in chronic gastric catarrh, especially that of alcoholic subjects. If the reaction is alkaline, the fluid usually consists of saliva that has been swallowed during the night and the vomitus consists largely of saliva in cases in which prolonged nausea has preceded the act of vomiting. If the reaction be acid the vomitus consists either of gastric fluid in excess—hypersecretion—or of food and mucus that have undergone acid fermentation. More commonly the vomited matter contains mucus and in some cases of acute and chronic gastric catarrh it is composed of masses of tenacious mucus. The vomiting of hyperacid gastric juice occurs in peptic ulcer of the stomach and in neurotic conditions, as migraine, hysteria, the gastric crises of tabes and exophthalmic goitre. In some cases of cholera the contents of the intestines are forced into the stomach and vomited, presenting the usual characters of the rice-water discharges and containing the comma bacilli of Koch.

Bilious Vomiting.—Bile is very commonly present, imparting a green or yellow color. It occurs after repeated or violent vomiting and is of no great diagnostic importance. The early vomiting of considerable amounts of bile occurs in some cases of peritonitis and intestinal obstruction.

Vomiting of Blood — Hæmatemesis — Gastrorrhagia. — This symptom occurs in a number of morbid conditions and is of great importance in diagnosis. The differential diagnosis between hæmatemesis and hæmoptysis has already been considered. The vomited blood may be bright red and fluid—a sign that it has remained in the stomach but a brief period; or it may consist of reddish or reddish-brown clots that have formed during a longer period; or finally it may present the appearance of coffee grounds, indicating that it has been subjected to the action of the gastric juice for a sufficient time to undergo partial digestion, with alteration of the hæmoglobin and destruction of the erythrocytes. In some instances a superficial resemblance to recent blood may be due to the presence of red wine or various reddish-colored fruits or the jellies or preserves made from them; in others altered blood—"coffee grounds"—may be suggested by the presence in the vomitus of coffee, cocoa, minute fragments of boiled or over-cooked meat, and certain drugs, as the preparations of bismuth and iron. As a rule these uncertainties may be settled by close inspection and an inquiry into the facts, but there are rare cases in which a chemical, microscopic, or spectroscopic examination may be necessary to determine the question. Bright red blood is usually vomited in considerable amounts and in association with small clots, while the altered blood which resembles coffee grounds is mixed with the vomitus in small quantities.

Not all blood ejected from the stomach is derived from the vessels of that organ. Blood is frequently swallowed and then vomited. In hæmoptysis a portion of the blood coughed up is often swallowed. Blood readily finds its way from the nasal chambers or pharynx into the stomach, especially when the patient is in the recumbent posture. The blood oozing from the bitten tongue in the epileptic paroxysm may be swallowed during the postepileptic stupor or the vomited blood may be derived from the vessels of the œsophagus. Blood may be swallowed by malingerers, who sometimes suck it from a wound made for the purpose in the mouth or upon the hand or forearm. In very rare instances infants vomit milk stained with blood derived from a fissured or ulcerated nipple.

Bleeding from the stomach occurs under various conditions, of which the following are important:

1. Circulatory Derangements.—Portal obstruction and the resulting passive hyperæmia of the gastric mucosa lead to hæmatemesis. This symptom therefore occurs in cirrhosis of the liver, in malignant and other tumors of the porta, and in adhesive pylophlebitis. Copious hæmatemesis, occurring in hepatic cirrhosis and terminating fatally, occasionally arises from rupture of the veins of an enlarged œsophageal plexus. The visceral congestions resulting from cardiac mural and valvular disease tend also to hemorrhage. Hæmatemesis is occasionally encountered in massive enlargement of the spleen.

2. Hæmic Disorders. — Hæmatemesis is frequently symptomatic of the grave anæmias. It occurs in pernicious anæmia, leukæmia, hæmophilia, scurvy, and purpura hæmorrhagica, in profound jaundice, and after extensive burns. It has been observed in phosphorus poisoning and in acute yellow atrophy of the liver.

3. The Infections.—Vomiting of blood is of occasional occurrence in epidemic influenza, typhus, relapsing fever, and dengue. It is a prominent event in some forms of pernicious malarial fever, malignant variola, and yellow fever. In the last the vomiting of altered blood—*black vomit*—is characteristic.

4. Traumatism.—Contusions of the epigastric region, as from a blow or kick, crushing, and other injuries, are sometimes followed by the vomiting of blood. The vomitus is often blood-streaked after prolonged or violent vomiting. Under this caption must be placed the direct injury to the gastric mucosa caused by the corrosive poisons, caustic alkalies, the mineral acids, arsenic, and the like.

5. Specific Anatomical Lesions of the Stomach. — Cancer is a common cause of gastric hemorrhage. The blood is usually dark and altered and rarely profuse, slight oozing, either continuous or frequently repeated, being the rule. Even more common is gastric ulcer. The blood is usually abundant, bright red, and fluid. Copious hæmatemesis is suggestive of ulcer. Free and even lethal bleeding may occur in superficial erosions, and profuse hemorrhage may come from the erosions of the gastric mucous membrane which sometimes occur after operations upon the abdomen and especially in cases in which the omentum has been wounded. In gastric and duodenal ulcer, especially the latter, the blood may not be vomited but is passed in the stools. Miliary aneurism is a rare cause of gastric hemorrhage. It is not common for death to result directly from the bleeding, which is often repeated from time to time. Anæmia, frequently of high grade, results. Syncope with or without general convulsions may immediately follow profuse hemorrhage. Hemiplegia and amaurosis, which may be followed by optic atrophy, are rare sequelæ.

6. Certain Nervous Affections.—Hæmatemesis is an occasional event in hysteria, and cases of gastric bleeding have occurred in apparently healthy individuals in the absence of any local or general condition to account for it, and without a second appearance. This symptom is comparatively infrequent in epilepsy and in general paresis, and Schiff and others have directed attention to it as a rare phenomenon in local cerebral disease. In the newborn it may occur as an isolated symptom or with hemorrhage from other mucous tracts.

7. Fatal gastric hemorrhage may result from the rupture of an aneurism of the aorta or its branches into the stomach. Under such circumstances death may occur from blood loss without vomiting, the stomach being distended with blood.

Fecal or Stercoraceous Vomiting.—This is a significant symptom in acute obstruction of the bowel. The anatomical condition may be strangulation, intussusception, volvulus, or abnormal contents. The last of these—fecal masses, biliary calculi, and enteroliths—may cause acute obstruction by the sudden shifting of their position. Vomiting comes on early and is

persistent. The vomitus consists at first of the contents of the stomach, then of bile or bile-stained material, and finally of a brownish or blackish fluid of a distinctly fecal odor. In this fluid masses of fecal matter may be present. Retroperistalsis not rarely occurs in peritonitis and in some cases stercoraceous vomiting is the result of a gastro-intestinal fistula. Chronic intestinal obstruction is not usually attended by this form of vomiting even when of high grade. In the terminal paroxysms, however, it may occur.

Purulent vomiting is rare and not usually dependent upon primary disease of the stomach; it may, however, occur in phlegmonous gastritis. The more common cause is perforative ulceration of the wall of the stomach in hepatic abscess or empyema.

Parasites in the Vomit.—The *Ascaris lumbricoides* occupies the upper part of the small intestine. From this position it finds its way readily into the stomach and is often ejected with the vomit. In rare cases the segments of tænia are present in vomited matter and the hooklets and fragments of echinococcus cysts have been observed; so also trichinellæ and the larvæ of insects.

The quantity of the vomitus depends upon the volume of the stomach contents and the intensity of the act of vomiting. Very significant is the retention vomiting of pyloric obstruction and the large fluid vomiting in gastrectasis from other causes.

The Odor.—The vomit is commonly sour-smelling and often intensely acid. It is ammoniacal in uræmia and fecal in acute intestinal obstruction and in some cases of peritonitis. The odor of the vomitus in poisoning is sometimes of great diagnostic importance. Striking examples are carbolic acid, the garlicky smell in phosphorus poisoning, that of bitter almonds in poisoning by hydrocyanic acid and nitrobenzole, the vinegar-like odor in poisoning by acetic acid, and the smell of ammonia; less significant are the odors of alcohol or laudanum.

The reaction is commonly acid. Where there is an excess of saliva, bile, or blood the reaction is alkaline. In hypersecretion the reaction is intensely acid and patients speak of their teeth being set on edge by the taste. In uræmia the reaction may be alkaline.

The taste of the vomitus is, according to the patients, commonly sour and when bile is present, bitter. Blood imparts a salty or sweetish taste.

DEFECATION.

Significance of Abnormal Defecation. — The indigestible parts of the food, with débris, bacterial masses, and secretions from the intestinal tract, pass slowly through the large intestine and reach the sigmoid flexure, in which they accumulate. As the semisolid or solid material passes into the rectum it stimulates the sensory nerves of that part of the intestine, giving rise to a peculiar sensation and desire to defecate. This material is retained in the rectum by the two sphincter muscles, the internal of which is a band of the circular layer of involuntary muscles of the rectum. Upon the passage of fecal matter into the rectum the internal sphincter passes into a condition of tonic contraction, the relaxation of which marks the beginning of the act of defecation. The internal sphincter is composed

of unstriped muscular fibre and receives its innervation from the sympathetic system and from the sacrospinal nerves. The external sphincter ani is made up of striated muscular fibres and is to a large extent under the control of the will. Upon intense rectal stimulus the voluntary control is overcome and this sphincter is also relaxed. The act of defecation is therefore in part voluntary and in part involuntary. The voluntary factor is made up of the inhibition of the external sphincter and the simultaneous action of the abdominal muscles, the diaphragm being contracted and the glottis closed. Pressure is thus exerted upon the abdominal and pelvic viscera, with the result that the contents of the descending colon and sigmoid flexure are forced into the rectum. This pressure is augmented by deep inspiration and fixation of the respiratory muscles. The involuntary factor consists in the contraction of the muscles of the rectum, in particular the circular layer, and the relaxation of the internal sphincter, in part the result of reflex stimulation from the lumbar cord and in part from automatic peristaltic movements. The action of defecation is essentially, however, an involuntary reflex, as is well seen in infants and in soporose states.

Under normal conditions the bowels move once a day, the act being, like sleep and the taking of food, of diurnal rhythmical recurrence. There are healthy individuals, however, in whom the rhythm is not diurnal, but at intervals of two or three days or exceptionally longer, and in whom efforts to bring about the diurnal movement by means of purgatives are followed by manifest derangements of health. The normal periodical movement of the bowels is maintained by the observation of a fixed hour for this function, and various derangements, especially constipation, result from the neglect of this rule.

It is important for the physician to inform himself as to the periodicity, frequency, and character of the bowel movements and in certain cases to inspect the stools. Departures from the normal in respect of this function relate to constipation, diarrhœa, tenesmus, painful defecation, fecal incontinence, and the character of the discharges.

CONSTIPATION.

Constipation—costiveness—infrequent or difficult evacuation of fæces; retention of fæces. This condition is of great and varied diagnostic importance. Its cause may be constitutional or intestinal. Very often several causes are associated.

The more important constitutional or general causes of constipation are:

1. Personal peculiarity: Sluggishness of the bowels is frequently an hereditary and family tendency. It is far more common in persons of dark than in those of fair complexion and is especially associated with the traits that constitute the bilious temperament.

2. Unhygienic habits, as want of proper exercise, the failure to observe regularity in the hour of defecation or to devote to the act sufficient time, irregularity or undue haste in meals and the eating of unwholesome food or of excessive quantities of food. From this point of view constipation is primarily not a condition of the body but a condition of the mind. On the

other hand too little food or a diet consisting largely of proteid substances or which contains a minimum of undigested residuum tends to constipation. It is obvious that a sufficient bulk of residual material is required to form the fecal mass and excite peristalsis. The insufficient ingestion of fluid tends also to cause constipation.

3. Dehydration of the tissues by profuse and frequently repeated sweating, diuresis from the action of drugs, the polyuria of diabetes insipidus and mellitus, or repeated hemorrhages is attended by constipation.

4. The febrile infections, except those in which diarrhœa is an especial symptom, are characterized by a tendency to constipation. Even in these affections constipation very often gives way in the later course of the attack to diarrhœa, and the latter may assume the guise of a critical discharge, as sometimes occurs in croupous pneumonia.

5. The habitual use of purgative drugs is a fruitful cause of constipation.

6. Constipation is a very common condition in the anæmias, especially in chlorosis, and is often a troublesome symptom in neurasthenia and hysteria.

7. General asthenia and cachectic states are very often attended by constipation; so also conditions in which the abdominal muscles are overdistended and their contraction hampered, as obesity, ascites, large abdominal tumors, and pregnancy.

Among local causes of constipation the following are to be considered:

1. Alterations in the quantity and quality of the intestinal juices and a deficiency of bile or pancreatic secretion. Under these circumstances constipation may be an important symptom of fever, chronic diseases of the gastro-intestinal tract, and diseases of the liver, biliary passages, and the pancreas. It is to be borne in mind that the normal presence of bile in the intestine constitutes a powerful stimulus to peristalsis.

2. The motor mechanism of the intestine may be at fault. The defect may be nervous, as in organic disease of the nervous system—myelitis, meningitis, and tetanus, or functional, as in hysteria and neurasthenia. Or the defective intestinal innervation may be the manifestation of a general asthenia. The arrest of peristalsis and tympanites in severe enteritis, some cases of appendicitis and in peritonitis and acute pancreatitis are primarily due to derangements of the nerve-supply to the bowel, secondarily to paresis of its muscular wall. Chronic intestinal catarrh and portal congestion from hepatic or cardiac disease are often attended by constipation due to impaired nutrition of the muscular coat of the bowel. Atony of the colon and especially of the muscular wall of the sigmoid flexure is an important local cause of constipation. Dilatation of the colon is attended with constipation. Large collections of scybala may accumulate in the sigmoid flexure and be felt upon palpation of the abdomen. Constipation due to this cause is encountered in neurasthenia and hysterical persons and is common in the insane. It occurs also in bed-ridden and elderly individuals.

3. Local disease of the rectum or anus or of adjacent organs is a common cause of constipation. When such conditions, as is usually the case, render the act of defecation painful, the patient is apt to postpone it unduly and there is very often reflex spasm of the sphincters which renders it for

the time impossible. Under these circumstances fecal material accumulates in the rectum and sigmoid flexure of the colon and greatly adds to the discomfort of the patient. Such local disorders are inflamed hemorrhoids, anal fissure, irritable ulcer, prostatic inflammation or abscess, and a tender retroverted uterus or prolapsed ovary.

4. Constipation is observed in malignant disease of the œsophagus, pylorus and bowel and in other chronic conditions in which a minimum of food is ingested or that which is taken cannot pass onward or is persistently vomited.

5. This symptom may be due to a contracted condition of the bowel—so-called spasmodic constipation. The narrowing of the bowel may be the result of ulcerative colitis or dysentery; a manifestation of hysteria or of the atrophic processes of advanced life. The bowel may be in a condition of permanent contraction or spasm at one part and dilated elsewhere. The stools are small and sausage-shaped or they may be liquid with hard scybalous masses varying in size from a marble to a walnut. Spasmodic constipation occurs in the pelvic disorders of women and in chronic lead poisoning.

6. Strangulated hernia is attended with acute constipation. Laxatives are without effect. There are vomiting and abdominal distention. Pain is usually present. Similar symptoms attend volvulus and other forms of intra-abdominal strangulation. In intussusception, the sausage-like tumor, tenesmus, bloody mucus, and a relaxed anus are significant. Acute retention of fæces with the signs of obstruction demands very careful and systematic examination of the abdomen, a digital exploration of the rectum, and examination of the hernial rings.

Chronic intestinal obstruction may be due to foreign bodies, very large gall-stones, tumors within the gut or exerting pressure upon its wall, masses of scybala, and strictures of every kind. The constipation is gradually developed; occasionally interrupted by watery diarrhœa and sometimes by attacks with the symptoms of acute obstruction. Three facts are of great importance: First, that fluid fecal matter may work its way past the obstruction from time to time; second, that the dilated and congested bowel below the obstruction may discharge a thin fecal-stained mucus; and, finally, that both these conditions are occasionally mistaken for diarrhœa.

7. Constipation in infants. Constipation in the new-born may be due to imperforate anus or a congenital stricture. In some cases it results from dilatation of the colon, which may attain enormous dimensions, or it may be due to simple atony of the large bowel.

Constipation in sucklings and especially in bottle-fed infants is often due to deficiency of the intestinal secretions, the fæces being dry and hard. This condition has been attributed to insufficient water and a deficiency of fat in the food. In older children attention to the hour of defecation and regular habits are as important as in later life. Constipation often results from enterocolitis, from impairment of the contractility of the muscular wall and derangement of the normal secretions. Acute constipation is frequently symptomatic of mechanical obstruction by foreign bodies, hardened and impacted fæces, twists, and intussusception.

Associated Symptoms.—Sensations of pressure and distention in the abdomen, uneasiness and pain, especially in the course of the transverse colon, loss of appetite, a furred tongue, a disagreeable taste, and uneasy precordial sensations are common. Patients very often attribute these phenomena to derangements of the liver or stomach. An effectual purge is of diagnostic importance. The results very often show that these symptoms are due to constipation.

Of especial importance are the morbid phenomena in the distribution of the hemorrhoidal veins that result from constipation. Pain before and after defecation, protrusion of the dilated blood-vessels, bleeding and the discharge of stringy mucus are common. Paroxysmal neuralgic pain referred to the coccyx or the suprapubic region or to the inner aspect of the thigh are less frequent. Gastric catarrh with manifold symptoms and occasional implication of the duodenum and bile passages also occurs. In some instances catarrhal jaundice results and in chronic constipation a slight icteric discoloration of the conjunctiva is often seen.

Constitutional derangements are not less common. They consist of headache, vertigo, depression of spirits, disinclination for work, and debility. Actual neurasthenia with the most varied and depressing symptoms may result from obstinate and prolonged constipation. It is on the other hand important to bear in mind that nervous disease is a frequent cause of constipation and that the most troublesome constipation may occur, for example, in hysteria. Under such circumstances a vicious circuit is established, the constipation aggravating and intensifying the symptoms of the disease of which it is itself a symptom.

The duration of constipation is largely dependent upon its cause. Simple forms resulting from neglect of hygienic laws may last three or four days; more troublesome cases may resist usual treatment for weeks. Stubborn constipation with severe symptoms suggests mechanical obstruction of the bowel. The passage of flatus is a favorable sign. In transient constipation the indican in the urine is not increased; in chronic obstruction it is apt to be increased.

DIARRHŒA.

Abnormal frequency and diminished consistence of the stools. This symptom is of the most varied significance. It results from increased peristalsis, particularly when the large intestine is affected, from diminished absorption of the contents of the bowel, from an excess of fluid in the bowel either in consequence of hypersecretion of the substances entering into the formation of the *succus entericus* or of transudation of serum, and in rare instances from direct abnormal communication between the stomach or small intestine and the colon.

Diarrhœa may therefore be symptomatic of deranged innervation of the bowel, mechanical or chemical irritation by its contents, the action of toxic substances, either in the bowel or in the blood-current, as in poisoning, autointoxication or the infections, defective nutrition or circulatory derangements of the wall of the bowel, or local disease, as ulceration or new growths in the bowel itself or adjacent organs.

Diarrhœa may be primary or secondary or it may be acute or chronic. The number of stools varies from 3 or 4 to 30 or more in the course of twenty-four hours, their consistency from semisolid to watery, and their color, odor, and other physical characters vary within equally wide ranges (see pp. 534, 535).

The recognition of the following forms of diarrhœa is essential:

1. Nervous diarrhœa. This symptom may denote mere increase of peristalsis in the absence of any lesion of the intestine, in hysteria, neurasthenia, the intestinal crises of tabes, exophthalmic goitre, Addison's disease, movable kidney, in the first dentition, and in emotional disturbances in healthy individuals of neurotic temperament. The characteristic manifestations of the underlying nervous disorder are of diagnostic importance. The stools are of gruel-like consistence and contain nothing of pathological importance. The attack begins abruptly and is of short duration.

2. Irritation of the intestine secondary to constitutional conditions. Diarrhœa may occur in uræmia, hyperpyrexia, extensive burns, sudden chilling of the surface, certain infectious conditions, as malaria and septicæmia, and as the result of the subcutaneous injection of such purgatives as podophyllin or magnesium sulphate. The urine should be examined in every case.

3. Increased intestinal fluid. The stools are abnormally frequent and watery after the administration of the hydragogue cathartics and in cholera nostras and Asiatica.

4. Irritation of the intestine by various ingesta, or pathological bowel contents. Abnormal peristalsis and looseness of the bowels is caused by indigestion, intestinal parasites, local fecal accumulations, poisoning by the salts of mercury, antimony, arsenic, copper and so forth, the purgative drugs, organic acids derived from the food or from its decomposition in the stomach or intestines, mushroom poisoning, unaccustomed or improper articles of diet, bulky or indigestible food, large quantities of cold food or drink, or the administration of enemata. In all cases the anamnesis and physical examination are of diagnostic importance.

5. Abnormal irritability of the bowel. Diarrhœa may be the manifestation of an idiosyncrasy, and is symptomatic of catarrhal inflammation and of ulcerative processes of all kinds, from superficial erosions from mechanical irritation to the specific ulcerations of enteric fever, dysentery, or tuberculosis.

6. Impaired absorption. Diarrhœa is not rarely due to extensive ulceration or atrophy of the mucosa, amyloid disease, and portal congestion. The diarrhœa of tabes mesenterica is largely due to failure in fat absorption.

7. Mucous colitis — membranous enterocolitis. This syndrome is characterized by paroxysmal diarrhœa accompanied by severe hypogastric or left iliac pain and the discharge of masses of mucus or membranous casts of the bowel. The attack occurs at varying intervals, and the disease is observed in neurotic persons, usually women.

8. Under very unusual circumstances a fistulous communication between the stomach or upper part of the intestine and the colon—usually its transverse part—may be the cause of diarrhœa with stools

containing undigested food and conversely of the eructation of intestinal gas or the vomiting of fecal material.

9. Lienteric diarrhœa. Normal stools are usually more or less homogeneous. They frequently, however, contain such indigestible articles as seeds, husks, the capsules of berries, fruit pits, and the like. The diarrhœa caused by excessive quantities of food or the ingestion of food that cannot be digested, or which attends forms of enteritis that interfere with normal digestion is characterized by the presence in the stools of undigested or only partially digested particles of food and is known as lienteric. Fragments of food may be recognized in the stools shortly after it has been eaten. This form of diarrhœa may be acute, as after errors in diet or acute enteritis, or chronic, as in chronic intestinal catarrh.

Associated Symptoms.—Diarrhœa is often unattended by any symptom other than the frequent recurrence of the peculiar sensation which invites to the closet. Usually there is uneasiness in the abdomen, which may be associated with local or general pain, often colicky, and tenderness. Severe diarrhœa is attended with thirst, appetite is impaired, and there is debility proportionate to the urgency of the intestinal symptoms. Local or general tympanitic distention of the bowel also occurs. Vomiting is common, especially in the diarrhœas of infancy. The loss of fluid not only causes thirst, but may give rise to faintness, collapse, cramps of the muscles, subnormal temperature, diminution of urine even to suppression and albuminuria. The acidity of the urine is increased and it gives the reaction for indican.

TENESMUS.

Rectal tenesmus—painful, ineffectual, and usually long-continued straining at stool. This symptom occurs alone, but it is very often associated with vesical tenesmus, partly because of the anatomical relationship of the organs, partly because of the common action of some of the causes. It consists of spasm of the musculature concerned in defecation and micturition. The violent spasmodic contractions are repeated at short intervals and are attended with such distress that in extreme cases children fall into general convulsions and adults faint. The discharge consists of small amounts of stringy, sometimes bloody, mucus from the anus or a few drops of urine as the case may be. Rectal tenesmus occurs in the course of irritating lesions of the rectum and anus, whether these be primary or secondary. It is a symptom of intussusception, dysentery, polypus, adenoma and malignant tumors of the rectum and sigmoid flexure, proctitis and periproctitis, hydatid cysts of the pelvis, mechanical injuries to the rectum by foreign bodies, or in exceptional cases in highly neurotic persons it may follow digital or instrumental examination. Tenesmus is not a common symptom of hemorrhoids or fissure of the anus. It may be caused by impacted fæces, masses of round worms, the presence of foreign bodies, and, in connection with vesical tenesmus, by stone in the bladder. It is also a distressing symptom in acute inflammation and abscess of the prostate gland. Tenesmus is easily recognized. Its cause may be obscure. When it is violent or persistent a digital or proctoscopic examination should be made under local or general anæsthesia.

PAINFUL DEFECATION.

The pain may be such as to cause fecal impaction from voluntary postponement of the act. The passage of a large hard fecal mass is attended with pain under normal conditions. In proctitis, inflamed hemorrhoids, fissure of the anus, prolapsus, irritable ulcer, and malignant disease of the rectum pain upon defecation is a conspicuous symptom. It is usually present in inflammation of the prostate and is sometimes symptomatic of acute inflammatory affections of the pelvic organs in women.

FECAL INCONTINENCE.

This symptom may be due to local causes, as laceration of the perineum involving the anal sphincters, surgical over-stretching, and malignant or syphilitic disease of the rectum; more commonly it is due to general conditions which profoundly affect the nervous system, as coma from any cause, myelitis and other diseases of the spinal cord, grave chorea, convulsive diseases, as epilepsy, tetanus, and strychnine poisoning, and certain severe infections, as enteric fever, dysentery, cholera Asiatica and nostras and cholera infantum. Involuntary discharges very often precede dissolution. The unclean habits of some forms of insanity cannot be placed in this group of symptoms.

THE GROSS PHYSICAL CHARACTERS OF THE STOOLS.

The fecal discharges of the healthy adult are of brownish color, cylindrical form, soft solid or semisolid consistency, 150 to 200 grammes in daily quantity, usually neutral or faintly alkaline in reaction when passed, and emit the offensive characteristic odor.

Abnormal variations in these respects constitute diagnostic criteria of some importance. The macroscopic examination is too often neglected. Laboratory investigation is sometimes necessary (see p. 229).

1. The color, which is due to the presence of altered bile pigment, principally hydrobilirubin, may be modified by certain articles of diet or by drugs. It may be rendered black by blueberries or by iron, manganese, or bismuth; yellow by rhubarb, colchicum, senna, or santonin; green by spinach or calomel or by certain chromatogenous bacteria. In sucklings and others who subsist upon an exclusive diet of milk the fæces are golden yellow or whitish; in those who live largely on meat they are brownish-black in color, and this is also the case with fecal matter long retained in the bowel as in obstruction. In jaundice due to obstruction they are grayish or putty-colored. When they are increased and thinned by intestinal hypersecretion or transudation their color is usually light brown or yellowish; when very watery, as in cholera, they are of a dirty-gray color—*rice-water discharges*. The presence of blood colors the stools red or black: red when the blood comes in considerable quantity under active peristalsis from the ileum as in enteric fever, or when it comes from the lower bowel as in dysentery, or from the rectum as in piles; black

when it is derived from the upper regions of the gastro-intestinal tract as in peptic ulcer, or when it is thoroughly mixed with the stool.

2. The form is lost in diarrhœa, the discharge being gruel-like or watery in consistence. The normal cylindrical or sausage-shaped stool may be modified in various conditions of the lower bowel. In prolapsus ani or stricture of the rectum, more rarely in intussusception, the diameter may be much narrowed—*pipe-stem stools;* in stricture or cancer of the rectum or the pressure of an enlarged prostate gland or abscess or in large pelvic tumors impinging upon the rectum the stools may be flattened or *ribbon-shaped;* in constipation from any cause, but especially that which results from atony and distention of the colon, they often assume the form of irregular, round, hard masses like the dung of sheep—*scybala.*

3. The consistence is increased in constipation. The fluid is resorbed and the mass tends to become hard and dry. The consistence is diminished in diarrhœa. Serous stools are observed in cholera Asiatica, cholera nostras and cholera infantum; in poisoning by antimony, arsenic, and mushrooms. Small, dribbling, serous stools occur in some cases of intestinal obstruction from cancer and other causes. Serous stools contain little or no fecal matter.

4. The quantity varies greatly. It is diminished when the diet is concentrated or consists principally of meat; increased when the diet is largely made up of starchy and vegetable foods. The amount voided at one effort depends of course upon the frequency of the act and may attain in cases of constipation as much as 1000 grammes. The quantity in diarrhœa is increased by the hypersecretion of the intestinal glands and the transudation of serum from the blood-vessels. In starvation the total quantity may not exceed 90 grammes a day.

5. The reaction and odor. The reaction is faintly acid in nursing infants and alkaline in some forms of intestinal fermentation. The acidity is due to carbohydrate fermentation or the presence of fatty acids. The reaction is of no great diagnostic value. Depending upon the amount of proteid decomposition and the putrefactive bacteria present, the odor of the stools is more or less offensive. Diets that allow much proteid residue to reach the large bowel usually give foul-smelling movements. A milk diet in health gives an almost odorless stool. Indol and skatol, derivatives of proteid decomposition, are mainly responsible for the characteristic fecal odor.

The odor in healthy infants is sour and unlike the fecal odor of the stools of adults. The so-called "albuminous decomposition" in the fæces of infants and the resulting putrid odor are due to the decomposition of the undigested proteid of the milk in the large intestine. In cholera infantum it is sometimes faintly musty, sometimes suggestive of the washings of meat. In the absence of bile the stools have a peculiarly offensive odor.

The presence of milk curds in the stools of infants indicates an error in the quantity or quality of the food and is one of the earlier symptoms of enterocolitis; the presence of curds in the stools of adults who are taking a milk diet, as in enteric fever, constitutes an indication for the use of alkalies in connection with the milk or a reduction in its quantity.

Abnormal Substances in the Stools.—In lienteric diarrhœa the stools contain undigested particles of food. Other abnormal substances are by no means uncommon and may be of great diagnostic importance. Among these are mucus, blood, pus, fat in excess, gall-stones, intestinal sand, concretions, intestinal parasites, sloughs, and foreign bodies that have been swallowed.

Mucus.—Minute particles of mucus may be observed upon the surface of the formed stools in health. Large quantities covering the stools or expelled with them in masses indicate a deranged secretion of the mucous glands of the colon or rectum. Masses of mucus that may be shaken out in water into sheets or tubular casts of the intestine are diagnostic of membranous colitis. Mucus intimately admixed with the fecal matter may come from the small bowel. Mucus in the stools is symptomatic of mechanical or pathological irritation of the bowel and is seen in such conditions as impacted fæces, foreign bodies, intestinal parasitism, new growths, intussusception, and all forms of dysentery, enterocolitis, and proctitis.

Blood.—A distinction is made between "hemorrhage from the bowel"—the discharge of red blood unmixed with fecal matter—and "melæna"—blood intimately mixed with the fæces and occurring in the form of "tarry" or pitch-like masses, usually of semisolid consistence and glistening appearance. The difference consists chiefly in the time the blood remains in the intestine and therefore in general terms indicates the portion of the gut into which it has been discharged. If, as in the case of peptic ulcer of the stomach or duodenum, the hemorrhage has been high up in the intestinal tract, the blood remains a considerable time in the bowel, and is subjected to mechanical conditions by which it is incorporated with the fecal contents, undergoing at the same time a sort of digestion by which its physical characters are much changed. If, on the other hand, the blood is poured out lower down in the bowel and under the influence of an active peristalsis is speedily evacuated, it maintains the characteristic appearance of fresh blood, often bright red and sometimes commingled with recently formed clots. The appearance of the evacuations therefore is of diagnostic importance in this respect. On the other hand, a copious hemorrhage from the ileum, as in enteric fever with active peristaltic movement, usually shows itself in the discharge from the bowel of bright red blood, while a slow oozing from the colon with tardy onward propulsion in the bowel may appear in the stools as "coffee-grounds" or even as "tarry" material.

Blood is frequently present in the stools in quantities so minute that its presence can only be detected by chemical examination—occult blood (Part II, p. 232).

The more important causes of blood in the stools are portal congestion, ulceration of the intestinal mucosa, neoplasmata and in particular malignant disease of the gut, intestinal parasites, embolism of the mesenteric arteries, intussusception, and traumatism.

1. *Portal Congestion.*—This occurs in cirrhosis of the liver, portal thrombosis, and dilatation of the hemorrhoidal veins—piles. The diagnosis of hemorrhoids rests upon the habitual or occasional discharge of bright red blood with the stools, the appearance and habits of the patient, and the signs obtained upon inspection or a digital examination.

2. *Ulcerative Processes in the Bowel.*—Intestinal hemorrhage occurring in the course of an attack of enteric fever is of positive diagnostic importance. It means the erosion of an arterial twig in an ulcer. If the hemorrhage is profuse it may at once lead to collapse with the associated symptoms of internal hemorrhage; if slight, the stools may be tarry or contain slight amounts of red blood without symptoms. In either case the appearance of blood in the stools is of prognostic importance, since it denotes deep ulceration which may be followed in a day or two by a more abundant blood loss or by perforation. Other ulcerative processes that lead to the appearance of blood in the stools are dysentery, syphilis, and tuberculosis. Under these conditions the blood appears in the form of streaks or stripes upon the stools or admixed with mucus or pus. Dysenteric stools may present the appearance of meat washings or of masses of blood commingled with liquid fecal matter. The mere presence of blood in the stools does not under ordinary circumstances justify a diagnosis. The anamnesis and a systematic investigation of the present condition of the patient are necessary.

3. *Malignant Disease of the Bowel.*—Blood in the stools is in many cases the first symptom to attract the attention of the patient to carcinoma. The stools are not, however, characteristic, and a systematic examination, which may reveal the presence of an abdominal tumor, is necessary. General failure of health, secondary anæmia, signs of intestinal obstruction, and cachectic phenomena are confirmative.

4. *Intestinal Parasites.*—The *Ankylostomum duodenale* is a common cause of persistent melæna among workers in the soil and miners. Griesinger first drew attention to this parasite as the cause of Egyptian chlorosis. The worms infest the upper portion of the small intestine and are very abundant in the jejunum. The diagnosis rests upon the prevalence of the condition among workmen in tunnels, brick-yards, excavations, and the like, and the presence of the ova in the stools.

5. *Embolism of the Mesenteric Arteries—Infarction of the Bowel.*—In consequence of valvular lesions of the heart, but with no great frequency, embolism of this distribution may occur. It is probable that the occlusion of small vessels produces no symptoms of importance and that the circulation may be reëstablished. If the superior mesenteric artery is occluded, or a large branch, the symptoms are sudden collapse, violent colicky pains, signs of peritonitis, and thin, blood-tinged stools or hemorrhage from the bowel.

6. *Intussusception.*—This affection occurs in infancy and childhood. Bloody stools are of diagnostic importance since they occur in at least sixty per cent. of the cases either spontaneously or after the administration of an enema. The blood is commonly mixed with mucus. Associated symptoms are tenesmus and a sausage-shaped tumor in the line of the colon. Vomiting and tympany are less common.

7. *Traumatism.*—Injuries of the bowel as a cause of bloody stools commonly involve the rectum, and when they do not penetrate the peritoneum may be readily overlooked. The abundant venous supply favors free bleeding, and, since the blood is often retained in consequence of spasm of the sphincters, the signs for a time may be simply those of inter-

nal hemorrhage and collapse. The anamnesis is of importance and a digital examination reveals the actual condition. The presence of foreign bodies may be thus discovered in children, idiots, and insane persons.

8. *Constitutional Conditions.*—Intestinal hemorrhage is occasionally symptomatic of leukæmia, hæmophilia, purpura hæmorrhagica, and scurvy. This symptom is, however, so closely associated with the general phenomena of those diseases that it is of secondary importance in their diagnosis.

9. *Miscellaneous Causes of Intestinal Hemorrhage.*—Bloody stools are of infrequent occurrence in consequence of the rupture of an aneurism of the abdominal aorta into the bowel, jaundice, acute yellow atrophy of the liver, phosphorus poisoning, yellow fever, pernicious malarial fever, and very rarely septicæmia.

Concealed Hemorrhage. — Concealed intestinal hemorrhage may occur in the foregoing conditions. If small it may give rise to no symptoms, although prolonged and unsuspected bleeding may be the cause of profound secondary anæmia with its usual symptoms; if large the hemorrhage, while not for a time appearing at the anus, occasions the symptoms of internal hemorrhage,—namely, collapse, restlessness, air-hunger, pallor, a pinched face, cold extremities, a rapid, weak, even imperceptible pulse, urgent thirst, and a tendency to syncope.

Pus.—In small quantities pus may be present in the stools in dysentery, enteritis, colitis, proctitis, and in ulceration of the colon or rectum due to malignant growths or syphilis. Small amounts of pus may be present in the stools in profuse leucorrhœa or urethritis; but under these circumstances its appearance is without diagnostic importance, since the associated symptoms will fully explain it. In large quantities and usually in single discharges, or in large quantities at irregular intervals, pus may be present in the stools in consequence of the rupture of an abscess, or the establishment of a fistulous communication between a purulent collection and the bowel. Such abscesses are usually periproctic, pelvic, or perinephric; sometimes appendicular; and, less commonly, in the gall-bladder, hepatic or infradiaphragmatic.

Fatty Stools.—The appearance of the discharges is greasy and glistening. An excess of neutral fat is present in obstructive jaundice and in various forms of pancreatic disease. Fatty diarrhœa, with icterus and sugar in the urine, has been observed in acute suppurative pancreatitis. Overfeeding and indigestion in infants may be the cause of fatty stools, and Biedert has described a fat diarrhœa in which the percentage of fat is enormously increased. The condition is primary in which the ingestion of fat is excessive and which may be corrected by modification of the food, and secondary which is due to catarrhal inflammation of the intestine or disease of the pancreas.

Gall-stones—Biliary Calculi. — Gall-stones have been found to be present in Europeans in from 5 to 10 per cent. In the East gall-stones are said to be extremely rare. Gall-stones vary in size from a concretion barely perceptible to the naked eye to the size of a walnut or larger. They are spherical, oval, or angular, the surface being smooth, mammillated, or faceted. When large they are commonly single; when small they may number hundreds. In a case of mine the small stones numbered by actual

count 300. When extremely small they are described as biliary or intestinal sand. Their color varies from a whitish-gray to dark yellow or brown, sometimes black. Their consistence is usually firm, but they are often friable, being crushed by pressure between the thumb and forefinger, with crystalline fracture. In some cases, however, they are extremely hard.

Intestinal Sand.—Small brown or green calculi, spherical or irregular in shape and of rough surface, and varying in size from grains of sand to small shot, are sometimes present in the stools in considerable quantity. This material may or may not be preceded by attacks of colic. These calculi are of variable composition. They consist in some instances of inorganic salts, as calcium carbonates and phosphates, magnesia and iron, together with organic matter, bacteria and urobilin. Cholesterin is not present. A nucleus may sometimes be demonstrated. It is formed of a grain of quartz sand or a minute particle of the case of a fruit seed. In other very rare cases calcium sulphate has been the chief constituent. This form of intestinal sand occurs in intestinal neuroses of the secretory type.

Pancreatic Calculi.—Kinnicutt has recently studied the subject of the discharge of pancreatic calculi during life. The decisive evidence of pancreatic lithiasis consists in the presence of the characteristic concretions in the stools. They are composed chiefly of calcium carbonate. They are extremely rare—a fact due in part to the small size of the calculi and their friability, so that they may be voided in fragments or particles not easily recognized.

Intestinal Concretions — Enteroliths. — Concretions of various kinds occur in the stools. They are comparatively rare. The following forms are encountered:

1. Hard round fecal masses—scybala. They occur in chronic constipation, especially in elderly people, and in cases in which after abdominal operation partial obstruction of the bowel occurs as the result of adhesions.

2. Enteroliths. Earthy concretions are sometimes observed in the stools. They are largely composed of magnesium phosphate, the alkaline carbonates, and organic matter. They are hard, dense, and made up of concentric layers about a chalky nucleus that very often surrounds a foreign body. They are usually oval and are very rarely, when several are present, faceted, and occur in early and middle life.

3. Concretions composed of vegetable fibres or of hairs that have been swallowed are light, porous, usually of irregular shape, and frequently show upon section open spaces or cavities in their substance. They are sometimes found in the cæcum and may attain the size of an orange. They are sometimes made up of the insufficiently ground husks of oats or the capsules of small fruits. They occur more commonly in early life and in females.

4. Certain drugs and similar substances, as chalk, magnesia, bismuth, and shellac, when taken in undue quantities, form intestinal concretions, which appear in the stools and reveal their true nature only upon chemical examination.

Intestinal concretions when of small size occasion no characteristic symptoms. When of larger size they may be arrested at a point of stenosis of the bowel, or upon the occurrence of contraction and œdematous swelling, and they may then give rise to the symptoms of intestinal obstruction. Large concretions are usually arrested in the cæcum, in the colon, or in the ampullæ of the rectum, less frequently above the ileocæcal valve. Obstruction in the upper part of the small intestine may be caused by concretions formed in the stomach or by gall-stones.

Intestinal Parasites. — The Ascaris lumbricoides — *round worm* — and its ova are frequently found in the stools of children and young adults. Oxyuris vermicularis — *thread-worm, pin-worm* — a very common parasite, infests the rectum and colon; intestinal cestodes — *tape-worms* — of which the common forms are the *Tœnia saginata* or *mediocanellata,* the *Tœnia solium,* and the *Bothriocephalus latus,* show themselves in the stools in the form of segments or proglottides, and their ova are usually present in great numbers (see p. 254, Vol. II).

Sloughs.—The invaginated portion of the bowel in intussusception may slough off *en masse* and be discharged from the bowel. Polypi of the intestine or rectum may also become detached by sloughing and be discharged with the fæces. Masses of necrotic tissue may become separated from malignant or other ulcerating growths in the intestine and be discharged with the fæces. They are to be distinguished from fragments of undigested meat. The intestinal sloughs in enteric fever may sometimes be recognized in the stools and are often mistaken for milk curds.

Foreign Bodies.—The most diverse articles may be found in the stools, having been swallowed by accident or design. Small articles of all kinds may be swallowed by children, idiots, and dements; bird-seed and the like by hysterical persons; coins, rings, and gems by professional thieves; nails, glass, fragments of china, etc., by fakirs, and such articles as artificial teeth or even a clinical thermometer by unconscious persons, and all of these things have been voided with the stools.

XI.

THE SKIN; PHYSIOLOGICAL AND PATHOLOGICAL CHANGES AND THEIR SIGNIFICANCE; ŒDEMA; SUPERFICIAL VASCULAR CHANGES; NAILS; HAIR.

THE SKIN.

Changes in the skin not only occur as manifestations of cutaneous affections but they also constitute important diagnostic signs of diseases of the internal organs. The methods of examination are inspection and palpation. The clothing is to be so arranged as to facilitate the necessary investigation.

The condition of the skin varies within physiological limits at different periods of life and in the sexes. In infancy and childhood the skin is distensible, elastic full, of fine texture, and faint rosy color. The capillary

circulation is active, pressure causes local pallor which quickly disappears. In middle life the skin is finer, softer, and shows more physiological turgescence in women than in men. With advancing age the skin loses its elasticity. Partly for this reason, partly on account of the diminished amount of subcutaneous fat, and partly because of the larger development of connective tissue, wrinkles develop. The skin in elderly persons is paler and more abundantly pigmented than in the young. The skin of very fat persons frequently has a disagreeable unctuous feel; it may be firm and tense or loose and flabby. The skin is sometimes flabby and relaxed in fat babies who are not properly fed. In the cachexias of infancy, such as that of congenital syphilis or marasmus, the skin is muddy, loose, inelastic, and sometimes wrinkled like that of old men.

Color.—The normal tint of the skin, the so-called flesh color, depends upon the blood showing through the upper layers of the integument and the epidermis. The changes in color are quantitative and qualitative, physiological and pathological. Quantitative changes consist in varying degrees of color, from blushing to blanching. They are best observed upon the face. On the other hand qualitative changes in the color of the skin are studied best upon other parts of the body where the flesh color is paler and less variable. The mucous membrane of the conjunctiva, lips, and mouth must always be examined.

Variations in the flesh color depend upon the amount of blood in the cutaneous vessels, the amount of the blood-coloring matter, that is, the percentage of hæmoglobin, and the thickness of the tissues covering the vessels. It is obvious that since any of these factors may vary in degree the quantitative changes in the color of the skin do not always have the same diagnostic significance.

Pallor.—The skin may be pale by reason of general or local deficiency of blood, that is to say, in consequence of anæmia or of contraction of the capillaries. The various forms of anæmia have in common a diminution in the coloring matter of the blood—*oligochromæmia*. Pallor, even when persistent, does not in all instances justify a diagnosis of anæmia, since there are many habitually pale persons whose blood shows upon examination a practically normal constitution both as regards the erythrocytes and the hæmoglobin. Many such individuals present no symptoms of constitutional or local disease and regard themselves as in perfect health. The pallor in these cases is due to an abnormality of the skin, either an unusual opaqueness of the superficial layers or a deficiency in the blood supply or a combination of these two conditions. If the conjunctival mucous membrane and that of the lips and mouth present a normal appearance, the pallor is due to the first of these anomalies. In the majority of instances, however, marked and persistent pallor is associated with other evidences of more or less decided derangement of health. Even under these circumstances in a certain proportion of the cases the blood shows no abnormal change. Two explanations of the pallor may be advanced: first, a reduction in the total quantity of the blood, which nevertheless retains its constituent elements in normal proportion; second, that the skin, particularly of the face, as the result of abnormal conditions of the circulation receives a diminished amount of blood. Since we have no

clinical method of determining the total volume of blood in the body, the first of these explanations is purely theoretical and without practical application. The second explanation finds support in the constant presence of other symptoms indicative of circulatory derangements, among which are a small and feeble pulse, general asthenia, over-filling of the superficial veins, slight cyanosis, faintness, and dizziness. The part played by enfeeblement of the heart's action on the one hand and by vasomotor derangements on the other cannot in all cases be satisfactorily determined. Lowered blood-pressure does not necessarily induce pallor, since in this condition the lumen of the peripheral vessels is widened and their contents increased; but diminished blood-pressure gives rise to pallor when the chief factor in its production is cardiac weakness and the vasomotor tonus is maintained. Increased vasomotor tonus may be the cause of pallor of high intensity. Among the more important diseases in which pallor occurs as the result of a diminution in the blood supply to the vessels of the face, without marked changes in the composition of the blood, are gastro-intestinal affections, both acute and chronic, diseases of the heart, pulmonary consumption and other chronic infections—conditions ultimately leading to anæmia which in many cases is profound. To this group the transient pallor of intense emotion, nausea, vertigo, syncope and collapse, in which vasomotor derangements and cardiac failure are associated in the production of lowered blood-pressure, bears a close etiological relation. Indoor occupations, dependence upon artificial light, mining and the like cause permanent pallor.

Clinically the following points are important: (a) Transient pallor is caused by cardiac failure, as in nausea, rigors, syncope, and shock, or by vasomotor spasm, as in the intense emotions of fright, fear, anger, in pain, epilepsy, and other paroxysmal neuroses. Transient pallor is frequently but not always followed by more or less intense flushing.

(b) Sudden and more persistent pallor accompanies hemorrhage, acute poisoning, and overwhelming infection—the malignant forms. Associated with other symptoms of collapse it is a striking and suggestive sign of internal hemorrhage, such as may occur in a large pulmonary cavity; as the result of the rupture of an aortic aneurism into the pericardial, pleural, or peritoneal sac; in consequence of a perforating lesion in peptic ulcer or enteric fever; in rupture of the sac in ectopic gestation, or in concealed uterine hemorrhage before or after parturition. Small hemorrhages do not necessarily cause pallor except when frequently repeated or persistent.

(c) Gradually developing pallor is a symptom of almost all serious acute and chronic diseases. In the acute infections it usually passes off with convalescence; in the chronic diseases its intensity is very often a measure of the gravity of the case. It is sometimes seen in altered conditions of living, as in the case of young immigrant girls who during the process of acclimatization not rarely permanently lose their color without changes in the blood or other signs of ill health. The pallor in persistent slight hemorrhage, such as occurs in neglected hemorrhoids, is very often intense, as is the pallor of chlorosis, pernicious anæmia, and the secondary anæmias which occur in chronic poisoning, chronic infections, chronic suppurative processes, nephritis, and valvular and mural disease of the heart.

Redness. — The change in the color of the face is quantitative. It is due to two causes: first, thinness and transparency of the superficial layers of the integument; second, increased fulness of the capillaries — *hyperæmia*. An abnormally high hæmoglobin percentage cannot of itself be regarded as a cause of the increased redness of the complexion. Whether or not a true plethora occurs is undecided. Physiologically we find the redness of the skin of the face greater in persons who live in the open air and are especially exposed to sunlight and the wind, which increase the cutaneous circulation. An abnormally transparent skin is the evident cause of the blooming redness of the cheeks occasionally seen in chlorotic girls— *chlorosis florida*. Very characteristic in these cases is the contrast between the color of the skin and the blue-white conjunctivæ. Among the physiological causes of intensification of the color of the skin are powerful muscular effort and the action of external heat, as in hot baths, friction of the surface, exposure to fire or heat, radiation from other sources, sunburn and the like. Extreme cold also produces cutaneous hyperæmia of the face. Habitual exposure to heat or cold, especially when associated with moisture, causes the chronic purplish hyperæmia of the hands frequently seen in washerwomen and bartenders who are otherwise in good health.

Transient reddening of the skin dependent upon vasomotor influences occurs under certain psychic influences, especially embarrassment and shame. The reddening of the skin in such cases is not restricted to the face but may spread over the throat and even the upper part of the chest. In these latter situations it may be irregularly distributed in such a way as to give rise to errors in diagnosis as regards actual disease of the skin, as erythema, especially in sensitive persons, and particularly in women when it is necessary to remove the clothing from the upper part of the body for the purposes of examination. One-sided flushing of the face occurs in certain forms of migraine and in affections of the cervical sympathetic.

In addition to the foregoing facts the flushing incident to pyrexia, certain infections, and the action of drugs deserves attention.

Fever.—The flushing of the skin in acute febrile conditions is very characteristic. It is often attended with slight turgescence and sometimes with a tendency to sweat. The flush of fever is usually widely distributed over the surface. It is more marked in young persons of fair complexion than in older persons and in brunettes. It has a tendency to localize itself in the cheeks where it is sometimes circumscribed or unilateral, as in croupous pneumonia. Circumscribed flushing of the cheeks in persons otherwise pallid is a very striking phenomenon in the hectic fever of advanced phthisis. In children the fever flush is sometimes so intense as to suggest the existence of erythema or scarlatina. In rare instances pyrexial flushing occurs during the first week of enteric fever, especially in young persons of fair skin, and may be so marked as to give rise for a time to uncertainty in diagnosis.

Tache cérébrale is a cutaneous vasomotor phenomenon which occurs especially in young persons in acute febrile affections, as cerebrospinal meningitis, enteric fever, and influenza, in certain functional nervous affections, as hysteria, neurasthenia, and sometimes in organic diseases of the brain and spinal cord. It is called forth by slight irritation of the

skin, such as is produced by tapping with the finger-tip or drawing the finger or a pencil smartly over the surface. A white spot or line appears and is shortly followed by a bright red discoloration which persists for several minutes.

Dermatographism.—This condition, closely allied to the above, is not uncommon in neurotic persons, particularly in those who suffer from urticaria. Wheals may be produced by drawing the finger or a pencil somewhat firmly over the surface. Letters and other symbols may be brought out in a conspicuous manner and often last for several hours. The itching characteristic of urticaria does not occur.

Drugs.—The reddening of the face caused by alcohol is of diagnostic importance. The expression "flushed with wine" is significant. The slightly turgid, purplish-red face of chronic alcoholism, with its distended venules, is unfortunately too familiar. The flush produced by the nitrites and especially by the inhalation of amyl nitrite resembles the blushing due to psychic causes. Flushing of the face follows the administration of certain poisons, as belladonna, opium, and hyoscyamus.

Cyanosis.—This term is used to designate the dusky blue or purplish color of the skin dependent upon the circulation in the capillaries of blood abnormally rich in carbon dioxide and poor in oxygen. Cyanosis may be general or local.

General cyanosis is dependent upon two factors, first, deficient oxygenation of the blood in the lungs, as the result of which the arterial blood reaches the capillaries containing less oxygen and darker in color than normal; second, stasis in the venous radicals, resulting in an accumulation of venous blood in the capillaries of the skin, which by the retardation in its flow becomes richer in carbon dioxide and darker in color. Since the conditions are universal it may be assumed that the bluish discoloration exists not only in the skin but in all the tissues of the body. Only in its intense forms does cyanosis show itself in all parts of the surface. When slight it appears in certain parts only and here it is in all instances more intense than elsewhere. These regions are the face and especially the cheeks, the tip of the nose, the ears, lips and mucous surface of the mouth, which have an especially abundant capillary circulation and translucent integument. Other points in which cyanosis is especially manifest are the hands and feet, particularly the terminal phalanges and the nails, in which blood stasis is favored by their remoteness from the heart.

The primary derangement may be respiratory or circulatory. The interdependence of the respiration and circulation is such, however, that when cyanosis is marked there is general derangement of both in varying proportion.

Respiratory.—All conditions which interfere with the respiratory function and thus reduce the aëration of the blood may give rise to cyanosis. They are comprised in four groups:

(a) All affections which interfere with the access of air to the vesicular structure of the lungs, such as retropharyngeal abscess, stenosis of the larynx caused by pseudomembranous exudate, as in diphtheria, œdema of the glottis, pseudocroup, laryngismus stridulus, pertussis, paralysis of the abductor muscles, tumors of the larynx, foreign bodies in the pharynx,

larynx, trachea, or bronchi, all forms of stenosis of the trachea, including thyroid enlargement and other deep-seated tumors of the neck, as well as mediastinal and other intrathoracic tumors, strangulation, bronchitis, and bronchial asthma.

(b) Affections which interfere with the action of the respiratory muscles, including paralysis and atrophy such as occur in bulbar paralysis and peripheral neuritis; spasmodic contraction of these muscles, as that of tetanus or epilepsy; painful affections, such as myalgia, pleurisy, and peritonitis, in which the respiratory movements are instinctively restrained; finally, the action of drugs, such as opium and its preparations, which depress the respiratory centres.

(c) Affections which diminish the respiratory surface. This group includes all forms of consolidation of the lung, croupous pneumonia, bronchopneumonia, including tuberculous infiltration and acute miliary tuberculosis, atelectasis, pressure atelectasis from pleural and pericardial effusion and pneumothorax. In emphysema the respiratory surface is not only greatly restricted but its functional integrity is also impaired.

(d) Conditions in which respiratory movements are restricted and the respiratory surface is circumscribed by subdiaphragmatic pressure, as in hydramnion, enormous ascites, enlargement of the liver or spleen, or massive abdominal or pelvic tumors.

Under all these circumstances the aëration of the blood in the lungs is diminished and venous stasis is favored by the reduction in the normal aspiratory function of the lungs which constitutes an important factor in the circulation. The absence of cyanosis, often observed in advanced phthisis with extensive destruction of the lungs and very limited respiratory movement, is probably due to the great wasting of the body and corresponding reduction in the mass of the blood, to the aëration of which the remaining limited vesicular structure is still adequate. Cyanosis is marked in proportion as the interference with respiration is rapid and urgent. In chronic cases the interference may reach a high grade without causing cyanosis during repose, though this symptom may appear upon slight exertion.

Circulatory.—Primary derangements of circulation which cause cyanosis may be referred to the following groups:

(a) Affections of the heart and arteries, including valvular disease with impaired or ruptured compensation, myocarditis, acute dilatation of the heart, the cardiovascular changes which occur in chronic nephritis, other forms of arteriosclerosis, and pericarditis.

In persistent foramen ovale and other forms of cardiac malformation, such as stenosis of the pulmonary artery, there is very often marked and continuous cyanosis. To this condition of congenital cyanosis the term *morbus cæruleus* has been given. In acquired conditions permitting an admixture of venous blood with arterial within the vessels, as in the very rare cases of aneurism of the aorta communicating with the vena cava, cyanosis is a suggestive symptom.

(b) Conditions affecting the pulmonary circulation. In disease of the mitral valve, both stenosis and insufficiency, even when compensation is good there may very often be seen, especially upon exertion, a slight degree of cyanosis. This is a manifestation of the changes caused by the habitual

increase of tension in the pulmonary circuit and the bronchial catarrh which to some degree is almost constantly present. Though having its primary cause in the circulatory apparatus this form of cyanosis must be looked upon as respiratory.

Pressure upon the pulmonary artery or veins by massive pericardial effusion, mediastinal tumor or aneurism is a very common cause of cyanosis. The circulation of the pulmonary capillaries is obstructed in many of the conditions involving the respiratory apparatus which give rise to cyanosis.

Blueness of the general surface, very often intense, is produced by overdoses of certain of the coal-tar derivatives, especially acetanilid, by nitrobenzole, and by poisoning with illuminating gas.

Local cyanosis results from venous stasis, from compression of the part or from venous thrombosis. Cyanosis of the head and neck or an upper extremity may result from the pressure of a tumor or aneurism upon the jugular, subclavian, innominate, or descending cava, the distribution of the cyanosis corresponding with the point of pressure. Similar cyanosis of one or both lower extremities may result from pressure involving iliac veins or the ascending vena cava or from venous thrombosis. Local venous thrombosis giving rise to cyanosis of an arm is sometimes seen in cancer of the breast with secondary implication of the axillary glands.

Cyanosis, often of high grade, results from vasomotor derangements. To this cause must be referred the cyanotic discoloration of the extremities and ears which follows exposure to intense cold, the cyanosis of paralyzed members, and the bluish discoloration of the hands which occurs in hysterical and neurasthenic persons. In the latter group of cases the local cyanosis is sometimes associated with œdema—the *blue œdema* of French authors.

Local cyanosis is seen in intense inflammation involving the skin.

The conditions which give rise to cyanosis, namely, retarded circulation and reduced oxygenation, interfere with the local production of animal heat. In cyanosis the skin and extremities show reduction of surface temperature.

Jaundice—Icterus.

These terms are used to designate the peculiar pathological yellow discoloration of the skin, mucous membranes, and fluids of the body caused by the circulation in the blood of bile pigment. The change is qualitative. There are two forms, obstructive and toxæmic.

Obstructive Jaundice.—This is the more common form. The discharge of bile into the intestine is interfered with wholly or in part by stenosis or closure of the bile passages. As a result there is resorption of the bile, the pigments of which discolor the tissues in shades varying from light yellow to a dark brownish-yellow or olive-green. The darker shades of jaundice result either from change of the original bile pigments to darker pigmentary bodies or from their excessive accumulation in the skin. The more intense and darker forms of jaundice occur in protracted cases. In permanent obstruction the color may be greenish-black or bronze—the so-called *black jaundice.*

Among the more important causes of obstructive jaundice are catarrhal inflammation of the mucous membrane of the duodenum or the common

duct; gall-stones and parasites, as the round worm, in the ducts; stricture or obliteration of the duct; tumors developing in the duct or exerting pressure upon its orifice; external pressure upon the duct by tumors of the liver, stomach, pancreas, kidney, or omentum, or by enlarged glands in the porta, or in rare instances by aneurism or fecal accumulation.

The yellow discoloration is observed first and, when slight, only in the conjunctivæ and the mucous membrane of the mouth. Its presence may be detected by pressure upon the mucous membrane of the everted lip with a glass slide, thus expressing the blood and permitting the yellow stain of the tissues to become apparent. It is sometimes distinct at certain pale areas of the hard palate. The slighter grades of icterus cannot be recognized in artificial light. Superficial resemblances to jaundice are seen in the dirty yellow or muddy discoloration of the malarial and malignant cachexias. In these conditions the absence of yellowness in the conjunctival and oral mucous membranes is conclusive. The collections of yellow subconjunctival fat occasionally seen in elderly persons are only in the most remote way suggestive of jaundice. The yellow discoloration which occurs in picric acid poisoning presents superficial resemblances to jaundice. The absence of bile pigment in the urine is important.

Pruritus is a troublesome symptom. It is usually more marked in the chronic cases. Lesions of the skin, the result of scratching, are not uncommon. Sweating is common and may be localized. Urticaria, furuncles, lichen, xanthelasma, and other diseases of the skin occur. In some of the chronic cases circumscribed patches of dilatation of the capillary vessels and minute arteries—telangiectasis—develop in the skin of the face and body and occasionally upon the mucous membranes. In protracted and severe cases there may be hemorrhages into the skin, usually in the form of purpuric spots upon the lower extremities, but sometimes as large ecchymoses, and in some instances spontaneous bleeding from the mucous membranes occurs. The blood in chronic jaundice coagulates very slowly—ten to twelve minutes, instead of about four in the case of normal blood—and troublesome and even fatal hemorrhage, usually in the form of uncontrollable capillary oozing, may follow operation or injury. The sweat is bile-stained and discolors the clothing. The urine contains bile pigment and may show the color reaction to Gmelin's test before the yellow tint appears in the mucous membranes or the skin. The color varies from light yellow with a greenish tinge to a deeply opaque black-green. In intense or long-standing jaundice the urine commonly contains albumin and tube casts which are bile-stained. Upon agitation the dark urine of jaundice is frothy and is often popularly compared to porter. The sputa are not often bile-stained, except when pneumonia is present. On the other hand the saliva very rarely shows the yellow discoloration, which is likewise absent in the tears and milk.

As no bile is discharged into the intestine the stools are of a pale drab or clay color. They are usually pasty and very fetid. The absence of bile in the fæces is of importance in the differential diagnosis between obstructive and toxæmic jaundice. Commonly there is constipation; occasionally diarrhœa. The pulse, in obstructive jaundice especially, in recent cases is usually slow and may fall to 30 or lower. The frequency of the respiration

is also diminished, in some instances to 10 or 8 per minute. The temperature may be subnormal. These symptoms are attributed to the action of the biliary salts, which undergo resorption together with the bile pigment. They are not constant and when present not necessarily unfavorable.

The patient is usually depressed and irritable. In severe cases melancholia may develop. The liability to the occurrence of the condition called cholæmia constitutes a serious danger in persistent jaundice. The patient falls into the so-called typhoid state, with fever, rapid pulse, dry tongue, and muttering delirium. Convulsions and coma develop and rapidly prove fatal. This group of symptoms resembles uræmia. They have been attributed to poisoning by cholesterin—cholesteræmia. The toxic substances have not been determined.

Toxæmic Jaundice.—The jaundice is associated with the presence of various poisons in the blood which act directly upon the red blood-corpuscles and in some cases upon the liver-cells. Among these poisons are (a) snake venom, phosphorus, arsenic, chloral hydrate, chloroform, and ether; (b) toxins elaborated within the organism in the course of the specific infectious diseases, as yellow fever, relapsing fever, malaria, pneumonia, enteric fever, typhus, and scarlatina; (c) the toxins of septic conditions, pyæmia, malignant endocarditis, acute yellow atrophy of the liver, Weil's disease, and epidemic jaundice. The symptoms are generally less intense than in obstructive jaundice. The discoloration of the skin is usually slight; exceptionally, as in the case of acute yellow atrophy and malignant jaundice, it is intense. The stools are colored with bile, sometimes deeply. The urine may be dark from increase in the normal urinary pigments but gives little or no reaction for bile pigment. Toxic jaundice of slight degree frequently appears during the course of febrile affections and under other circumstances and may be without unfavorable prognostic significance. On the other hand in many cases the conditions in which this form of jaundice occurs are attended with profound constitutional disturbance, manifest in intense fever, delirium, suppression of urine, hemorrhages into the skin and from mucous surfaces, convulsions and coma, and very often terminate in death.

The jaundice due to obstructive changes in the bile passages was formerly spoken of as *hepatogenous;* toxæmic jaundice as *hæmatogenous.* Concerning the mode of origin of toxæmic jaundice there is much diversity of opinion and the cases differ among themselves. In groups of cases there is probable resorption of bile pigments from the liver as the result of pathological processes involving the finer ducts or the liver parenchyma itself. Some pathologists attribute the icterus, so common in pneumonia, to a catarrh of the finer bile passages dependent upon venous stasis, while others attribute it in part at least to the interference with the respiratory movement of the diaphragm caused by the consolidation of the lung, and resulting in an accumulation in the smaller ducts of bile which undergoes resorption. The rapid course and profound disorganization of the liver in acute atrophy and in phosphorus poisoning suggest the possibility that other forms of grave toxæmic jaundice may be due to as yet unknown parenchymatous changes in the liver. On the other hand most of the poisons

which cause icterus exert a destructive influence upon the erythrocytes. It has been shown experimentally, however, that the yellow pigment in poisoning by certain substances, as toluylendiamine, is not formed in the blood but in the liver, the hæmoglobin being transformed into biliary pigment in that organ. As a result of this transformation the bile pigments accumulate in the liver in such quantity that they cannot be wholly excreted, a certain portion undergoing resorption. In consequence of these facts the term *hæmatohepatogenous* has been suggested for this form of jaundice. In the present state of knowledge the etiological designation toxæmic jaundice is to be preferred. The term toxæmic-obstructive jaundice has been suggested by Hunter.

Normal and Abnormal Pigmentation—Melanoderma.—The physiological pigmentation of the skin shows wide variations not only in different races but in different individuals of the same race. Among the fair-skinned a blonde and a brunette type are recognized. The latter is characterized by a darker color of the hair, skin, and iris. Normally the skin is more deeply pigmented in the exposed portions of the body to which the light and air have free access than elsewhere; upon extensor than upon flexor surfaces in the region of the joints; and about the nipples, linea alba, and genital organs. During pregnancy the pigmentation in these latter situations is greatly increased, especially in brunettes, and upon the face and in other portions of the body there are occasionally seen irregular, abnormally pigmented areas known as *chloasma gravidarum—masque des femmes enceintes.* Patchy pigmentation of the skin is a common symptom of uterine disease. In sedentary persons of constipated habit irregular patchy pigmentation of the skin is common, especially about the face and eyes.

Freckles or ephelides are another physiological pigmentation of the skin without diagnostic importance. The pigmentation appears in circumscribed spots varying from one to several millimetres in diameter, chiefly upon the face, but also in other parts of the body, especially the backs of the hands and arms. They are more common in fair than dark persons and are almost always present in individuals with red hair. The spots are more abundant and the pigmentation deeper in summer than in winter, when they sometimes wholly disappear.

The pigmentation following measles and showing the characteristic form and arrangement of the eruption is not wholly without interest to the clinician, and the localized pigmentation which follows the application of sinapisms and blisters deserves passing mention.

The vagabond's skin is a term applied to the diffuse pigmentation resulting from lousiness and dirt and the scratching caused by these conditions. The pigmentation sometimes reaches a very high grade. It may be arranged in a very characteristic manner in stripes corresponding to the lines of scratching. This condition has been confounded with the pigmentation of Addison's disease.

Melanosarcoma, especially when generalized, very often produces a deep and widespread cutaneous pigmentation. Under these circumstances in exceptional cases the urine also contains abnormal pigment.

In advanced pulmonary tuberculosis a striking brownish discoloration of the face or the whole body is sometimes observed.

In ABDOMINAL NEW GROWTHS, especially cancer or lymphoma, diffuse cutaneous pigmentation occasionally occurs. It is not uncommon in tuberculosis of the peritoneum.

In HÆMACHROMATOSIS, such as occurs in hypertrophic cirrhosis, diabetes, and other conditions, pigmentation of the skin may be present.

EXOPHTHALMIC GOITRE may be associated with abnormal pigmentation.

GASTRIC.—In rare instances diffuse pigmentation attends gastric ulcer and dilatation.

In SCLERODERMA cutaneous pigmentation may be general and of high grade.

CARDIAC.—In chronic disease of the heart and arteriosclerosis diffuse pigmentation may occur.

ADDISON'S DISEASE.—The bronze discoloration of this affection is clinically the most important form of abnormal pigmentation of the skin. It usually shows itself first upon exposed surfaces, as the hands and face, and is more intense in those regions in which the skin is normally more deeply colored than elsewhere. It begins as a faint smoke-gray discoloration and progressively deepens to an intense bronze or mulatto hue. In the diffuse smoky coloration isolated intense dark brown points may be distinguished. The grayish pigment patches seen upon the mucous membrane of the mouth are characteristic of Addison's disease. The palms and soles as well as the nails commonly remain pigment free. The discoloration of Addison's disease may suggest intense jaundice, but the general condition, the yellow staining of the conjunctivæ and the mucous membrane of the mouth, and the presence of bile pigment in the urine are of positive diagnostic importance.

HEPATIC DISEASE.—The peculiar discoloration of the skin occasionally seen in cirrhosis and other diseases of the liver demands consideration. The color is a dirty brownish-gray. It is to be differentiated from icterus by the color itself, the absence of staining of the mucous membranes, and the condition of the urine. This pigmentation is of especial interest in connection with the bronzing of the skin that occurs in certain cases of diabetes—*diabète bronzé*—developing late in hæmachromatosis and associated with pigmentary cirrhosis of the liver and pancreas. The color suggests Addison's disease, but the presence of grape sugar, the physical signs of hepatic cirrhosis without jaundice, and the absence of the characteristic symptoms of Addison's disease are of diagnostic importance.

ARSENOMELANOSIS.—The pigmentation of the skin produced by the prolonged administration of arsenic in full doses sometimes presents a very close resemblance to Addison's disease. In a majority of the cases it entirely disappears when the drug is withheld; exceptionally it is persistent. It is important to know that in some instances the pigmentation of the skin has followed the use of arsenic in moderate doses.

ARGYRIA.—The prolonged administration of silver nitrate results in the deposition of particles of metallic silver or its albuminate in the internal organs and in the skin. The resulting discoloration is a peculiar bluish-gray which is more intense upon the face and hands and is not changed by pressure. The discoloration may be observed in the mucous membrane of the mouth. It is persistent and not amenable to treatment.

Albinism is a term used to designate developmental deficiency of pigment. In albinos the skin, hair, and eyes are conspicuous by the absence of pigment. The affection may be partial or universal. It is frequently associated with other developmental defects, especially coloboma. Nystagmus is common.

Vitiligo is a condition of the skin characterized by deficiency of pigment. The patches are usually circumscribed, very often distinctly marginate, and sometimes surrounded by a zone of pigmentation slightly deeper than normal. It may occur on any part of the body, but is common on the back of the neck and shoulders, the abdomen, and scrotum. There are no subjective symptoms. It occurs in adolescents and young adults.

Leucoderma or pigment atrophy, usually circumscribed or irregularly distributed, is encountered in exophthalmic goitre, myxœdema, scleroderma, and other constitutional disturbances.

Moisture.—There are wide variations in the activity of the sweat-glands within physiological limits. Perspiration is excited by those causes which determine an active blood supply to the skin. It is therefore more abundant in warm weather, after exercise, hot baths, and hot drinks. An outburst of sweating may occur in connection with sudden intense emotion. A pathological increase of perspiration is termed hyperidrosis; its absence anidrosis. These terms are commonly used to designate conditions in which the increase or absence are persistent or habitual.

Hyperidrosis.—Free perspiration attends certain febrile diseases, especially rheumatic fever, some cases of enteric fever, acute polyneuritis, miliary fever, and septic conditions. A critical decline of fever, whether spontaneous or the result of the administration of antipyretics, is almost always attended by more or less abundant sweating. Perspiration is one of the processes by which, both physiologically and pathologically, the temperature of the body is lowered. Profuse sweating attends the crisis in pneumonia, relapsing fever, and typhus. Sweating is often abundant toward the close of enteric fever when the temperature curve assumes a distinctly remittent or intermittent type. The fall of temperature in the ague paroxysm is almost always attended with copious sweating. That of the hectic fever of phthisis and other wasting diseases usually occurs during the night or toward morning. It is attended with abundant sweating—*night-sweats*—and is of unfavorable prognostic significance. Profuse sweating occurs in some cases of phthisis in the absence of fever. Sudden abundant sweats are accompanied by sensations of great weakness and prostration which are in part due to the relaxation of the vessels following the sudden withdrawal of fluid. Excessive sweating occurs in the convalescence from some diseases. It occurs in collapse, urgent dyspnœa, and sometimes accompanies severe paroxysms of pain. In rare instances of diabetes abundant perspirations have alternated with polyuria. Increased sweating sometimes attends the suppression of urine that occurs in certain forms of nephritis. Under these circumstances crystals of urea may accumulate upon the skin and especially upon the face.

Localized sweating is not uncommon in pathological conditions. Hyperidrosis of the hands and feet occasionally occurs in neurotic individuals and sometimes in persons otherwise healthy. The condition is very

annoying. The sweat is usually copious and foul-smelling. Axillary sweating is an annoying constitutional peculiarity. Sweating of the head, especially during sleep, is an important symptom in rickets. Unilateral sweating of the head or face occurs in certain nervous diseases, as migraine and neuralgia, and may result from pressure upon the sympathetic by a thoracic aneurism or mediastinal tumor. Localized sweating depends upon vasomotor derangements. Diaphoresis follows the administration of certain drugs, especially ammonium acetate, pilocarpine, and many of the coal-tar derivatives.

Anidrosis.—Abnormal dryness of the skin occurs under conditions in which an excess of fluid is withdrawn from the body by way of its internal surfaces, or very little water reaches the blood by way of the gastro-intestinal tract—for example, profuse diarrhœa, continuous vomiting, diabetes mellitus and insipidus, chronic nephritis with polyuria, and the deprivation of fluid. The dry skin of myxœdema and general anasarca is largely attributable to the interference with the cutaneous circulation resulting from tension.

Modifications in the Perspiration.—Perspiration when abundant usually has a peculiar acid odor. That in rheumatic fever is acid and ill-smelling; the sweat of the hands, feet, and axilla is almost always foul; that in certain forms of nephritis has a urinous odor. The sweat may be discolored—*chromidrosis*—yellow from biliary pigments in jaundice; blue from the action of the Bacillus pyocyaneus. There are instances recorded of the sweating of a blood-stained fluid or blood in hysterical females—*hæmatidrosis*—and there exists a term—*menidrosis*—to describe vicarious menstruation by way of the skin. These conditions are of no importance in diagnosis. Various colored perspiration-stains upon the linen are not to be mistaken for instances of chromidrosis. It may prevent error to call attention to the fact that some of the aniline dyes undergo more or less marked changes in color under the action of perspiration.

Fulness of the Skin—Turgor.—The normal appearance of fulness of the skin is due to the blood and lymph in its vascular and lymph spaces. It varies in different individuals and in different parts of the body, and is more pronounced in females. In connection with an abundant panniculus it has much to do in causing the condition described by the French as *embonpoint*. Increased fulness of the skin is seen in fever and other conditions attended by active cutaneous circulation; decreased fulness in all conditions in which the cutaneous circulation is diminished without stasis, particularly in emaciation, the cachexias, and under the deprivation of fluid. Increased fulness is manifested by rounding of the contours, especially those of the face, and usually by a deeper color of the skin, while diminished fulness produces accentuation of the angles and is usually associated with more or less pallor. In the former condition the skin is smooth, soft, and elastic; when pinched up into folds it rapidly reassumes its normal surface. In the latter such folds only slowly disappear. Normal fulness or turgor is to be distinguished from œdema and anasarca by the pathological amount of fluid in the skin in the latter, the loss of the normal cutaneous elasticity, and by the persistence of the pitting made by pressure of the finger. The difference between "looking well" and "looking bad"

very often depends upon slight transient variations in the normal fulness of the face, which is diminished in conditions of exhaustion and depression and increased after repose and in pleasurable excitement. The turgor of the skin is usually increased in exophthalmic goitre. Greatly diminished fulness of the skin such as occurs in ileus, peritonitis, cholera, and some cases of shock, and which precedes death, gives rise to the *facies Hippocratica* seen in these conditions.

FIG. 201*a*.—Œdema in chronic parenchymatous nephritis.—Jefferson Hospital.

Œdema—Dropsy.—An abnormal accumulation of serous fluid collects in the lymph spaces of the skin and the subcutaneous connective tissue as the result of a disturbance of the balance between the fluid which transudes from the capillaries and that which is taken up by the lymphatics. This disturbance of balance may be due to (a) venous obstruction, (b) altered condition of the blood —*hydræmia*, (c) inflammation, and (d) œdema of nervous origin. The diagnostic significance of œdema depends upon its location, extent, and mode of development and its causal relations to local or constitutional diseases. General œdema is described under the term *anasarca*. The skin is distended and the normal surface landmarks obliterated. When œdema is marked the surface is tense, pallid, and glistening. In rapidly developing recent œdema it has a translucent appearance. In some surfaces, especially upon the abdomen and thighs, transparent parallel stripes appear, similar to those seen on the abdomen in pregnancy. These are due to the collection of the fluid in the lines of separation of the distended tissues or in the enlarged lymphatic spaces. They usually disappear upon the subsidence of the œdema without leaving traces. Occasionally they leave permanent irregular linear scars. In œdema of high grade, especially under the influence of irritation or slight traumatism of the skin, blebs may form upon the epidermis which rupture

FIG. 201*b*.—Œdema of the legs with cutaneous blebs in a case of subacute parenchymatous nephritis.—Jefferson Hospital.

and are followed by the discharge of serous fluid. Occasionally, especially upon the legs and ankles, transudation of the fluid takes place through minute openings of the skin without bleb formation. Under these circumstances infection may occur, giving rise to erysipelatous or other inflammation. The pale color of the skin in œdema is caused by diminished capillary circulation from compression. The œdematous parts are sometimes cyanosed and in inflammatory œdema the skin is reddened.

The normal elasticity of the skin is impaired by tension and the inhibition of fluid. Pressure upon the œdematous part gives rise to pitting which only slowly disappears. Where the skin is normally distensible and elastic the pitting is more transient. This is especially the case in children. In

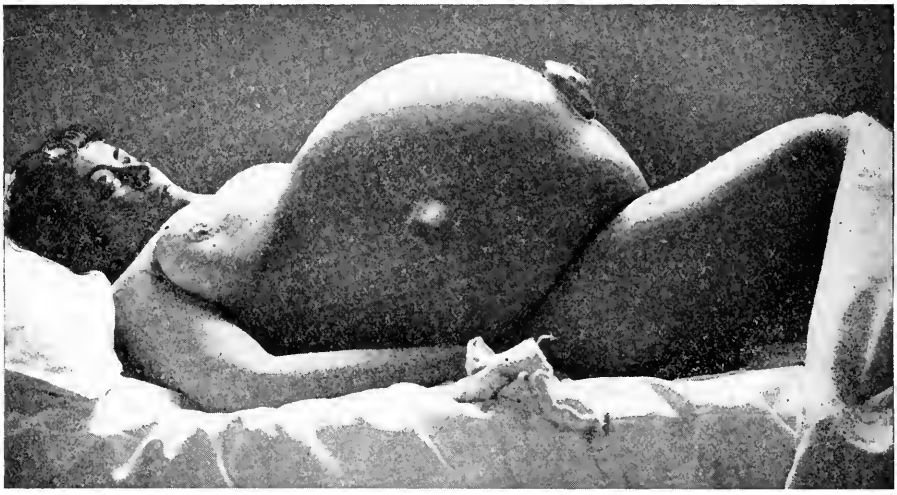


FIG. 202.—Œdema of abdominal wall and thighs in ascites due to atrophic cirrhosis of the liver.—Jefferson Hospital.

moderate œdema of long standing a gradual increase in the subcutaneous connective tissue develops and pitting is less marked and more transient.

(a) **Venous Obstruction.**—Factors in the production of this form of œdema are diminished general muscular activity, impaired pumping action of the organs of respiration, diminution of the aspiratory force of the heart in diastole and positive pressure on the veins. Coincidently the return flow of the lymph which is brought about by the same forces that maintain the venous circulation is impeded. This form of dropsy is frequently associated with effusion into the great serous sacs. The fluid which collects is clear, usually colorless, of low specific gravity, fibrin free, and contains a slightly smaller amount of proteids than the blood-serum. It is to be distinguished from an inflammatory exudate which is often turbid, sometimes bloody, of high specific gravity, and usually contains masses of fibrin. Changes in the tissues and particularly in the endothelium of the lymph-spaces also play an important part in œdema-formation—so-called "vital secretory" processes.

The collection of serous fluid in the pericardium is known as *hydroperi-*

cardium, in the pleural cavity as *hydrothorax,* in the peritoneal cavity as *hydroperitoneum* or *ascites,* in the brain as *hydrocephalus,* in the joints as *hydrarthrosis.* Any of the affections of the heart and lungs which, by interfering with the return of the venous blood, cause cyanosis may also cause œdema. Cyanosis and œdema are therefore frequently associated. This form of œdema appears earliest and reaches its fullest development in those regions in which the circulation, by reason of remoteness from the heart and the influence of gravity, is less active, as in the extremities and the lumbar regions and other dependent portions in bedridden patients. The face at first is free and becomes œdematous only when the anasarca reaches a high grade. Gravity plays an important part in the localization of the œdema. Œdema of the legs and feet while the patient is in the upright position may alternate with œdema of the back and thighs when he is in the recumbent posture. The patient who is apparently free from œdema while in bed may show œdema of the feet and ankles when he first rises. In prolonged maintenance of the lateral decubitus the œdema is more marked upon the dependent side. In anasarca of high grade, partly on account of their dependent position and partly on account of the distensibility of the skin, the penis and scrotum and the labia majora become enormously swollen.

Fig. 203.—Œdema of left leg due to a thrombus in the external iliac vein.—German Hospital.

Local œdema may be due to the obstruction of a venous trunk by thrombosis or pressure. Œdema of the arm from the pressure of enlarged axillary lymphatics upon the veins, and the œdema of the leg in thrombosis of the femoral vein are familiar examples. Obstructive œdema of the lower extremities is frequently secondary to peritoneal effusion, such as results from cirrhosis or portal thrombosis or from chronic peritonitis. The accumulation of the fluid presses upon the inferior vena cava or the common iliac veins. In other cases the œdema of the lower extremities and the peritoneal effusion are due to the same cause. When, upon investigation, the signs of peritoneal effusion are found to have preceded the œdema of the limbs, the latter condition is usually secondary.

(b) **Altered Condition of the Blood—Hydræmia.**—A watery condition of the blood is a common cause of œdema and other forms of dropsy. To this condition may be referred those forms of œdema which occur in nephritis, chronic wasting diseases, the anæmias, and cachexias. Not infrequently

associated cardiovascular disorders are present which interfere with the venous circulation, and in such cases the œdema from venous obstruction and the œdema of hydræmia are combined. This form of œdema differs markedly from the œdema of venous obstruction in its early localization, which is dependent much less upon remoteness from the heart and the action of gravity and much more upon the peculiarities of the lymph structures. It is characteristic of the œdema of certain forms of nephritis that it first appears in the face and especially about the eyelids. With this early œdema of the face pretibial œdema is often associated and is sometimes present in cases of nephritis, especially the chronic interstitial forms in which facial œdema is slight or absent altogether. The œdema of acute nephritis often develops rapidly and reaches a very high grade. Not infrequently it is associated with effusion into the serous sacs. In that form of nephritis characterized by contraction of the kidney œdema is very often slight in amount and a late manifestation, first showing itself when the hypertrophied heart begins to fail. In the subacute and chronic forms of parenchymatous nephritis the œdema is usually moderate, showing, however, temporary increases which accompany exacerbations of the disease. In the hydræmia resulting from large or frequently repeated hemorrhage, œdema is often pronounced. Œdema of the feet and ankles is a very unfavorable symptom in pulmonary consumption. Occurring in the absence of renal disease or especially in the absence of conditions giving rise to venous obstruction it is commonly an indication of approaching death.

(c) **Inflammatory Œdema.**—The local œdema in the region of inflammatory and suppurative processes is of diagnostic importance. It is sometimes known as collateral œdema. The color of the surface varies from a faint blush to a deep, mottled, cyanotic, purplish red. It is due to obstruction of the lymph circulation by the inflammatory exudate. In some instances it appears to be caused by an accumulation of the fluid part of the exudate in the tissues surrounding the inflammatory focus. It occurs in the region behind the ear in mastoid disease; about the angle of the jaw in mumps and parotid bubo; at the base of the thorax in empyema. It is an important sign of hepatic abscess, acute suppurative gall-bladder disease, and is sometimes seen in the right lower quadrant of the abdomen in appendicular abscess. It constitutes the so-called collar of brawn in severe anginose scarlatina.

(d) **Œdema of Nervous Origin.**—The rare cases of sudden transitory œdema of the face and neck, sometimes associated with symptoms of œdema of the respiratory or gastro-intestinal mucous membranes, must be ascribed to angioneurotic derangements. The mechanism which causes it remains unknown. The condition known as angioneurotic œdema is characterized by the sudden occurrence of local œdematous swellings of transient duration upon the face, hands, and elsewhere. Forms of localized œdema, described under the term giant urticaria, are of angioneurotic origin. The acute œdema associated with urticaria and gastro-intestinal crises which occurs in severe purpura, and the cases of œdematous swelling and tumefaction of the whole arm upon exertion, are to be referred to this group. The local œdema occurring as a symptom in peripheral multiple neuritis and the œdema of beriberi are probably of nervous origin, as is hysterical œdema.

(e) **Œdema due to Other Causes.**—Œdema neonatorum is a rare condition sometimes confused with sclerema, from which, however, it is pathologically distinct. It is encountered in feeble infants, especially those born prematurely or exposed to cold after birth. Cases of hereditary œdema have been described. The œdema is congenital and persistent; it involves one or both legs and is dense and inelastic. It shows no disposition to increase and is unattended by special inconvenience.

The œdema which occurs in trichiniasis is of diagnostic importance. It appears in the face and over the affected muscles, and undergoes remarkable fluctuations in degree during the course of the disease.

General œdema in the absence of nephritis is not infrequently observed in certain of the infectious diseases, as scarlet fever and diphtheria; it may follow the therapeutic injection of the different sera and in some instances the administration of potassium iodide. Slight œdema of the feet and ankles—a mere puffiness—is not uncommon in individuals otherwise healthy, after prolonged standing or walking or after forced marches.

Lymphœdema.—The transudation of lymph through the walls of the lymphatic vessels, or distention of the lymph spaces from mechanical obstruction, may cause great swelling, which is usually local or confined to a single limb. It results from pressure upon, or internal occlusion of, a lymph-vessel and is seen in the lymph scrotum and certain forms of elephantiasis caused by the Filaria sanguinis hominis and accompanied by chyluria. Lymphœdema involving a member—*macromelia*—sometimes occurs in lymphadenoma. This form differs from ordinary œdema by its greater firmness and brawniness—a very important point in differential diagnosis.

It is of diagnostic importance to recognize the distinction between the various forms of œdema and myxœdema—an affection of the thyroid gland characterized by swelling of the skin, eyelids, and other parts of the body, due to the deposition in the skin and subcutaneous tissues of a mucinous material. The skin is dry, rough, and swollen, but firm and inelastic, and does not pit on pressure.

Certain connective-tissue dystrophies present a superficial resemblance to localized œdema. The swellings usually involve the outer or posterior aspect of the extremities, but may appear at various parts of the trunk. They are to be differentiated from œdema by their localization, the absence of pitting upon pressure, and by other appearances of the skin characteristic of œdema.

Scleroderma, a brawny induration of the skin, in some instances suggests chronic œdema. Two forms are recognized, the circumscribed and the diffuse in which large areas are involved. The skin is brawny, hard, and inelastic. When circumscribed the patches are irregularly oval and vary in diameter; they may be as large as the hand. They are preceded by hyperæmia of the skin. The disease is more common in women than in men and frequently shows itself about the neck and breasts. The diffuse form involves the extremities and face. The skin is hard and firm with stiffness and tension. It is adherent to the underlying tissues and cannot be pinched up into folds. There is impairment of movement. Very often there are vasomotor disturbances with cyanosis. Pigment alterations are frequent—both melanoderma and leucoderma.

Sclerema neonatorum is a rare disease of the new-born in which the skin rapidly assumes the clinical appearance of scleroderma. It is usually fatal. It presents superficial points of resemblance to œdema neonatorum, from which it is to be distinguished by the complete absence of the ordinary signs of anasarca.

Scurvy sclerosis—a deep brawny infiltration of the subcutaneous tissues and muscles, with hemorrhagic discoloration of the overlying skin—frequently seen on the calves of the legs, is not to be confounded with œdema, although it is very often associated with it.

Subcutaneous Emphysema.—The presence of gas, usually air, in the meshes of the subcutaneous tissue gives rise to swelling and puffiness of the surface which may be either general or local. The appearance is not unlike that of œdema, but upon palpation a peculiar crackling is to be felt and heard, due to the displacement of bubbles of air in the tissues. The surface resistance is lower than normal and pitting from pressure does not occur. Upon percussion the sound is tympanitic. The skin is pale and has a distended appearance. In very rare cases subcutaneous emphysema is due to the presence of aërogenous bacteria—Bacillus aërogenes capsulatus—and allied organisms. This gaseous and necrotic œdema occurs in serious wound infection and may extensively involve the subcutaneous tissues of the body. The infection may proceed from the uterus, gastro-intestinal canal, or respiratory tract. Analogous to this condition is the subcutaneous emphysema of malignant œdema. It is sometimes associated with tetanus.

In the greater number of cases the air finds its way under the skin through an external wound or through the ulceration or laceration of some air-containing organ. Subcutaneous emphysema is, therefore, an accident of carcinomatous or other ulceration of the œsophagus, of diseases attended by violent paroxysmal cough by which the alveolar tissue is mechanically torn, or occasionally of the after-treatment of tracheotomy, the air being forced into the subcutaneous tissues by efforts of cough. The air usually accumulates about the root of the neck and over the manubrium. It may invade the tissues underlying the skin very extensively and sometimes involves the greater part of the body. As a rule it undergoes rapid resorption.

Cutaneous hemorrhages appear as spots or streaks of varying size, at first red, but quickly becoming darker. Small hemorrhages—*petechiæ*—frequently have their origin in the hair follicles. Larger hemorrhages—*ecchymoses*—are diffuse. Hemorrhages arranged in the skin in the form of lines and streaks are called *vibices*. The term *suggillation* is sometimes used to describe the ecchymosis following a bruise. *Hæmatoma* is a tumor containing effused blood. Cutaneous hemorrhages may occur upon any part of the body, but when due to constitutional disease they are more abundant upon the lower extremities. In consequence of transformations in the hæmoglobin the color during resorption undergoes progressive changes to blue, green, and yellow, and gradually fades. The appearance and distribution of petechiæ is characteristic of cutaneous hemorrhage. They are not usually elevated above the skin. Occasionally in purpura there are vesicular points distended with blood. In doubtful cases cutaneous hemorrhages may be distinguished from local hyperæmia or erythema by the fact that they do not disappear when the skin is made tense by traction

upon it of the thumb and finger or by pressure with a glass slide. In local hyperæmia the spot of redness disappears; in hemorrhage, owing to the expression of the blood from the surrounding capillaries, it becomes more distinct. Affections characterized by the extravasation of blood into the skin are collectively described under the term *purpura*.

Cutaneous hemorrhage is in all cases of diagnostic importance. The more important conditions with which it is associated are traumatism, intense venous stasis, the severe and especially the malignant infections, sepsis of various kinds, deep jaundice, and cachectic and anæmic states. It is a characteristic phenomenon of the action of certain snake venoms and under exceptional circumstances follows the administration of copaiba, quinine, ergot, iodine, and other drugs.

Hemorrhage into the skin occasionally occurs in acute myelitis, severe neuralgia, and in tabes. In the last it is very often transient. The bleeding points or stigmata that have attracted so much attention in rare cases of hysteria are of nervous origin.

It is frequently associated with arthritis. The relationship of these conditions has been regarded without adequate reason as rheumatic.

Other changes in the skin of diagnostic importance are: striations, desquamation, furunculosis, cicatrices, and glossy skin.

Striations.—The striæ of the skin of the abdomen and those occurring in œdema and peritoneal effusion, which resemble the striations of pregnancy, have already been described. They frequently disappear after resorption of the fluid, but may persist for a long time. Similar striations may attend the rapid development and equally rapid resorption of a thick panniculus adiposus. They are encountered in cases of great abdominal distention from rapidly developing tumors or other cause.

Desquamation.—Shedding of the epidermis is of diagnostic importance. A diffuse desquamation of the trunk and extremities, usually in the form of fine scales, occurs in the cachexia associated with emaciation. A similar fine-scaled desquamation follows measles. A coarser desquamation, sometimes lamellar, is almost constant after scarlet fever. A coarse desquamation follows erysipelas. The decrustation of the variolous diseases may be mentioned in this connection.

Furunculosis.—Boils or furuncles are the expression of an acute inflammation of a hair follicle and its sebaceous gland and the connective tissue immediately surrounding them. It is a local process due to an infection through the follicle by pus-producing organisms, usually the Staphylococcus aureus. Furunculosis occurs in conditions of lowered vitality, as during the convalescence from infectious diseases, especially enteric fever. The occurrence of furuncles in crops, or their persistent recurrence, is a common event in diabetes mellitus and should always lead to an examination of the urine for the presence of sugar.

Cicatrices or Scars.—These, whether recent or old, constitute important diagnostic signs. In doubtful cases special significance attaches to the presence or absence of the scars of vaccination and their characters and to the scars of smallpox. The scars of furuncles and carbuncles, of lupus, of inguinal buboes, and those left by tuberculous glands which have healed spontaneously or been removed are very suggestive in doubtful

cases. The scars left by tuberculous disease of the glands or bones are usually retracted and adherent. The presence or absence of scars upon the genitalia following the primary syphilitic infection is of great importance. They are usually difficult to discover in the female and are not always persistent. The serpiginous cicatrices of late syphilis cannot be mistaken. Cicatrices produced by therapeutic measures, such as cupping, venesection, leeching, the application of croton oil and tartar emetic ointment, and those left by surgical operations are of importance in the anamnesis. Occasionally scars upon the head or elsewhere constitute suggestive diagnostic evidence in obscure nervous diseases. Scars upon the tongue, the result of laceration during the epileptic paroxysm, may serve to clear up any doubt as to the character of convulsive seizures.

Fig. 204.— Distended veins of the leg and abdomen in a case of mediastinal tumor.—Jefferson Hospital.

Glossy Skin. — The appearance is characteristic. The skin is atrophied and attached to the subjacent structures. It is smooth, tense, and hairless and occurs most frequently and is more pronounced in the hands and fingers. It may develop elsewhere. The condition is the result of the trophic disturbance caused by traumatic or other lesions of the nerves. It is encountered in extremities that have been splinted after fracture, forms of neuritis, in conditions giving rise to the claw hand, in long-standing œdema, and in some advanced cases of arthritis deformans. It is not often seen in young persons.

Collateral Circulation in the Skin. —Dilatation of the superficial vessels frequently sheds light on symptoms dependent upon deeper circulatory derangements. In aged persons the overfilled veins of the extremities, showing prominently through the translucent atrophic skin, are an indication of the diminished cardiac power associated with general involution of the muscular system. The veins are darker in color than the blood which they contain —a phenomenon doubtless due to intensification of the color in transmission through the skin.

In tumors of the mediastinum which compress the great veins of the thorax, especially the venæ cavæ superior and inferior, the venous collaterals upon the anterior surface of the chest may be greatly enlarged. The blood is transferred from the compressed vena cava inferior by way of the intercostal veins and the internal mammary vein to the superior vena cava, or the reverse.

Thrombosis of the vena cava ascendens or of both common iliac veins results in the development upon the surface of the abdomen and anterolateral aspects of the chest of prominent sinuous venous enlargements, sometimes reaching the thickness of a finger, by which the blood from the lower extremities and the kidneys is conveyed to the veins of the thorax. In cirrhosis of the liver and portal thrombosis the compensatory circulation is often by way of the superficial veins. Occasionally a greatly enlarged para-umbilical vein passes from the hilus of the liver along the course of the round ligament and joins the epigastric veins at the navel, producing a large varix with wavy radial distribution of the veins known as the *caput Medusæ*. More commonly branches pass in the round and suspensory ligaments and unite with the epigastric and mammary systems. The vessels are numerous and of no great size. An important point of difference between the enlargement of the superficial collateral veins in obstruction of the vena cava and portal obstruction is to be found in their distribution. In the former the enlarged collaterals usually occupy the anterolateral aspect of the chest; in the latter the region around the navel and ensiform cartilage. It is important to determine in which direction the blood in the distended vein flows. This is done by emptying the vein by stroking it between two fingers and determining by removal of the pressure of the fingers alternately from which direction the blood stream comes. In great distention of the veins the valves become inadequate and this investigation is without result. The small vascular dendrites so often seen in irregular arrangement at the base of the thorax in chronic affections of the lungs and pleura indicate local areas in which collateral circulation has been established between the lungs and skin. They are especially common in pleural adhesions and are frequently seen upon the upper part of the back in chronic pulmonary tuberculosis with great pleural thickening. In many cases these minute dendritic enlargements at the base of the chest and the borders of the area of superficial cardiac dulness are without pathological significance, since they occur in healthy individuals. They have a certain clinical interest, however, since by their distribution they indicate upon inspection the position of the borders of the lung. Enlargement of the veins of the legs may be due to changes in their

Fig. 205.1.—Varicose veins.—German Hospital.

walls on the one hand or to thrombosis or pressure on the other. Enlargement of the veins of both legs is caused by the obstruction of the vena cava or both iliacs. Great enlargement of the veins of the legs, with the formation of varices, sometimes occurs after repeated pregnancies, and enormous varicosity of one leg, with great dilatation, frequently results from venous thrombosis following pregnancy or the infectious diseases, especially enteric fever. The varicosities which occur in the absence of pressure or thrombosis are largely due to changes in the walls of the veins themselves.

THE NAILS.

The appearance of the nails is to some extent indicative of the state of nutrition and habits. The deformity arising from biting the nails is characteristic and consists in shortening of the nail with projection of the tip of the finger, into which the edge of the nail tends to bury itself. Coarse longitudinal striæ associated with brittleness are said to indicate gouty tendencies. Small white flecks—*leucopathia unguis*—are the result of trifling knocks; the color is due to the presence of air among the cells. Transverse arched bands, dull and opaque, contrasting with the normal glistening surface, are seen after severe acute illness and indicate a period of malnutrition. They appear at the root of the nail and gradually advance. They are often seen after enteric fever and sometimes, in the case of relapse, there is a corresponding secondary band. Pressure upon the nails drives blood from the capillaries of the bed. The blanching is marked and somewhat prolonged in anæmic states. The nail is normally of a pink tint. Cyanosis shows itself early in the nails and their blueness is a measure of its intensity. When the capillary pulse is present it may be seen in the nail-beds, especially after slight pressure. The nutrition of the nails is affected in various skin diseases. They become dry, fragile, and malformed in neuritis, syringomyelia, Raynaud's disease, and scleroderma. Destruction of the nails occurs in the neuritis of Morvan's disease and leprosy. In hemiplegia and infantile palsy the growth of the nails upon the paralyzed side is retarded. *Onychia* is ulceration of the nail matrix. It may be due to syphilis or tuberculosis. In chronic disease of the chest the nails become hypertrophied and incurvated and the terminal phalanges clubbed—the *Hippocratic fingers*. These changes are seen most frequently in bronchiectasis and empyema, less often in phthisis. The deformity may develop very rapidly. Trifling lesions at the root of the nail—a mere splitting of the fold of epidermis at the side of the finger-nail, may be the point of serious infection. Malignant endocarditis and tetanus have arisen from this cause, and such sores upon the finger of the surgeon are frequently the seat of the initial lesion of syphilis. Congenital absence and deformities of the nails are not common. They may be hereditary and are usually associated with defects in development of the hair and teeth.

Shedding of the nails sometimes occurs in syphilis, alopecia areata, saccharine diabetes, hysteria, and other neurotic conditions. Extravasation of the blood beneath the nails may occur from injury or very rarely in purpuric affections. The blood-clot brings about a separation of the nail from its bed and its ultimate detachment.

THE HAIR.

Wide variations in color, texture, and abundance occur in different individuals. Certain changes are of diagnostic importance.

Color.—Grayness or canities may begin early in life. It is a sign of old age but there are people who grow old without growing gray. Gray hair in young people is sometimes hereditary. It is often associated with early arteriocapillary sclerosis. In a family in which nearly every member for three generations was the victim of chronic nephritis it was characteristic for the hair to turn gray before thirty. Early grayness, however, is not incompatible with excellent health. In rare instances rapid whitening of the hair has been attributed to extreme terror or anxiety. Circumscribed patches of gray hair are occasionally seen in healthy individuals. Their development sometimes appears to depend upon severe neuralgia involving the distribution of the supra-orbital branch of the fifth nerve. It is easy for the close observer to detect bleached or dyed hair. To the physician the former is suggestive of an undisciplined life, the latter of chronic lead poisoning as the cause of nervous symptoms otherwise obscure. Discoloration of the hair occurs in workers in copper, cobalt, indigo, and from local contact with dyes. Change in color may occur after severe illness with temporary loss of hair or after frequently repeated excessive sweating such as follows the hypodermic use of pilocarpine.

Hypertrichosis.—A growth of hair that is abnormal in quantity or in location may be congenital or acquired. It is a deformity rather than a disease. Very rare instances have been reported in which a growth of hair has covered the entire body except the palms, soles, terminal phalanges, upper eyelids, borders of the lips, prepuce, and glans penis. Hirsuties is more commonly localized. The causes of this condition are obscure. Among them heredity and irregularities or arrest of the sexual functions are prominent. A luxuriant growth of deeply pigmented hair has been observed in Addison's disease.

Atrophy of the hair occurs as the result of systemic conditions interfering with nutrition. The hair becomes dry and lustreless and splits at the end. It may undergo atrophy in local diseases of the scalp and in general conditions, as extreme emaciation and cachexia. Imperfect nutrition of the hair is conspicuous in myxœdema and occurs in advanced cases of pulmonary consumption.

Alopecia may involve the scalp or other hairy parts of the body. It may be congenital and is usually accompanied by defects in the teeth and nails. This form is very often hereditary. The hair does not usually grow in scars upon the scalp. Alopecia senilis accompanies other senile changes. Premature falling of the hair sometimes appears to be an idiopathic condition. It occurs in various local and systemic diseases. Among the latter are acute febrile infections, syphilis, and erysipelas. As a rule the hair grows again.

Alopecia areata or circumscribed patches of baldness appears in some instances to be a trophoneurosis occurring after shock or injury to the nervous system. In others it appears to be a local parasitic disease.

Diseases of the skin as such do not fall within the scope of this work. The cutaneous manifestations of the individual constitutional and organic diseases are considered elsewhere (see Part IV, Vol. II).

XII.

GENITO-URINARY SYSTEM; MICTURITION; THE REPRODUCTIVE ORGANS.

The diagnostic significance of the results of examination of the urine by laboratory methods is set forth in a previous chapter. The clinical facts may properly be considered separately.

MICTURITION—URINATION.

The urine is secreted continuously and conveyed by the ureters to the bladder, from which it is ejected at intervals through the urethra by the act of micturition. The urine accumulating in the bladder is prevented from escaping by the elasticity of the parts surrounding the internal urethral orifice and the contraction of the internal sphincter. When the accumulation reaches a certain point the desire to pass water is aroused. The external sphincter may be controlled by voluntary effort. The act of micturition consists in strong contraction of the bladder with the simultaneous relaxation of the sphincters and the contraction of the abdominal muscles, especially toward the close of the act. The contraction of these muscles with closure of the glottis and fixation of the diaphragm increases the pressure upon the contents of the abdominal and pelvic cavities and favors the complete emptying of the bladder. The peculiar sensation caused by the accumulation of urine in the bladder is followed by the reflex muscular contractions which constitute the act of micturition. Not only is this act largely under the control of the will, but the ability to void small quantities is also to some extent voluntary.

The average total daily quantity of urine in healthy men is 1200 to 1700 c.c.; in women the amount is less by 200–300 c.c. This represents the water excreted by the kidneys, but there is in health as well as in disease a vicarious relationship between the function of those organs and the skin and lungs, so that during increased cutaneous and respiratory activity, as in prolonged exercise or in warm weather, the quantity of urine may be reduced to 400–500 c.c. in twenty-four hours.

The quantity is diminished in disease in a corresponding manner. Thus, the loss of fluid by pathological sweating, profuse vomiting, colliquative diarrhœa, and hemorrhage is attended by more or less marked reduction in the urine. The quantity is also reduced in acute nephritis, in lowering of the blood-pressure from any cause, in many febrile conditions, and in dropsies and effusions into the serous sacs. Suppression of urine more or less complete is designated *anuria*—to a less extent *oliguria*.

An abnormal and continued increase of the daily quantity of urine, not accounted for by increased ingestion of fluid, constitutes the pathological condition designated *polyuria*. This condition occurs in diabetes insipidus and mellitus, in emotional states, in hysteria, during the epileptic paroxysm, in irritable lesions of the floor of the fourth ventricle, under the

influence of diuretics, in contracted kidneys, in chronic parenchymatous nephritis, in lardaceous renal disease, from increased blood-pressure, and as a result of the resorption of transudates and exudates.

The daily quantity is voided in several acts of micturition, usually about five, but the number within normal limits is largely determined by the habits and circumstances of the individual.

The following abnormal conditions are of diagnostic importance:

(a) **Dysuria.**—This term is comprehensively employed to designate difficult, slow, and frequent micturition, and, since these symptoms are mostly though not always attended with distress which is often urgent, it includes painful micturition.

1. **Vesical tenesmus** constitutes the most severe form of dysuria. It consists of painful spasm of the bladder and is often associated with rectal tenesmus. The spasm is often so urgent that the patient is unable to remain at rest, but returns at short intervals to the ineffectual and agonizing attempt to pass water, with the result that a few drops at most are voided with violent bearing down and burning pain in the urethra.

2. **Strangury.**—Not rarely a few drops of blood or bloody mucus are discharged in the spasmodic efforts at urination, and the condition is described as strangury. This term is, however, frequently employed interchangeably with tenesmus.

Very concentrated and acid urine is a cause of dysuria and the ingestion of certain condiments in excess, as mustard, pepper, and horseradish, may produce similar inconvenience. The absorption of cantharides and turpentine applied to the surface, or overdoses of these substances, may be followed by strangury. A sudden attack of vesical tenesmus for which no obvious cause is discoverable may be found upon investigation of the facts of the case to be a tabetic crisis.

Dysuria, especially these more intense forms, is liable to occur in almost all acute inflammatory diseases of the urinary tract. They are encountered therefore in posterior gonorrhœa and in gonorrhœal inflammation of the neck of the bladder, and in acute cystitis, prostatitis, and pyelitis. Dysuria also accompanies the chronic forms of these affections but is much less urgent and distressing. Tenesmus is also symptomatic of direct irritation of the bladder, as by stone, gravel, foreign bodies, parasites, and local ulceration. Reflex dysuria with tenesmus is sometimes present in renal colic.

Dysuria is a symptom of incomplete retention. When after the act of micturition there is residual urine, it is evident that the capacity of the organ will be more speedily reached than when it is emptied normally. Urination becomes more frequent and more difficult. This form of dysuria occurs in paresis of the bladder, as in tabes, hypertrophied prostate, tumor involving the neck of the bladder, stricture and phimosis, prostatic abscess, arteriosclerosis of the vesical arteries, spasm of the neck of the bladder, and œdematous swelling of the urethral mucosa in acute gonorrhœa. A chancre of the urethra may act in the same way, and in the variolous diseases pocks in the meatus may occasion similar symptoms.

Dysuria frequently attends general peritonitis, acute inflammatory diseases of the pelvic organs, and may occur in dysmenorrhœa.

(b) **Frequent Micturition.**—This occurs in polyuria. It is obvious that in the absence of dilatation of the bladder an increase in the quantity of urine must be followed by an increase in the frequency with which it is voided. Hence in diabetes insipidus and diabetes mellitus, in contracted kidneys and in some forms of pyelitis the frequency of urination is greatly increased. A diabetic who voids 6 litres of urine in twenty-four hours, with an average vesical capacity of about 300 c.c., would be obliged to pass water at least twenty times in the course of the day—a requirement which is slightly diminished by a gradual increase in the size of the bladder. When, on the other hand, the bladder has undergone concentric hypertrophy in consequence of chronic cystitis and its capacity is greatly diminished, the necessity to void urine at short intervals becomes imperative. If the call be not obeyed, as in deep sleep, the urine may be involuntarily discharged.

Frequent micturition is often due to psychical causes, among them fright and excitement. Soldiers in battle and students awaiting examination constitute oft-quoted examples. The urine is voided at short intervals and in small amounts and often involuntarily. It is also a common symptom in hysteria and neurasthenia.

(c) **Slow Micturition.**—The act is slow, prolonged, and difficult in all conditions characterized by mechanical obstruction to the outflow and in nervous affections attended by paresis of the vesical wall. Hence the form of dysuria encountered in incomplete urinary retention from any cause is characterized by slow or prolonged micturition. *Stillicidium urinæ* or the slow discharge of urine drop by drop has been described under the term "incontinence of retention." It occurs in the low fevers and in soporose and comatose conditions, when, because of the neglect of a routine physical examination and of the use of the catheter, the bladder has been allowed to become overdistended.

(d) **Incontinence of Urine.**—This condition is due to mechanical and nervous causes.

1. Mechanical causes are chiefly operative in women. Laceration of the perineum or injuries to the urethra sustained in parturition, relaxation of the floor of the pelvis, and cystocele are common causes of urinary incontinence. The urine may dribble constantly or be discharged in gushes upon any muscular effort which increases the pelvic pressure, as lifting, stooping, or coughing. Violent sudden cough, as in pertussis, may cause incontinence in depressed or asthenic conditions with relaxation of the sphincter muscles.

2. The nervous causes of incontinence are much more common. They may be cerebral, as in coma and shock, idiocy and dementia, or the stuporous states of the profound infections; spinal, as in traumatism, hemorrhage, and tumors of the cord, transverse myelitis, meningitis, and tabes; or reflex in consequence of the local irritation of ascarides, phimosis, contracted urinary meatus, stone in the bladder, cystitis, or highly concentrated or acid urine. To the last of these causes may be referred enuresis nocturna, which occurs in neurotic children and acquires the force of a morbid habit, the urine being voided involuntarily, as a rule during sleep, but frequently during the waking hours under excitement or preoccupation. If the vesical centre in the lumbar cord is destroyed, complete paralysis will ensue, with retention or the dribbling incontinence of retention.

(e) **Retention of Urine.**—Retention and incontinence are very constantly associated, and are due in many instances to the same causes. Thus, retention may occur in coma from any cause, in the soporose states incident to profound toxæmia, as in the graver forms of the infectious diseases and especially in the terminal infections, in peritonitis, in acute pelvic inflammations, and in injuries and diseases of the spinal cord.

Temporary loss of vesical power sometimes results from overdistention in consequence of prolonged voluntary retention. Mechanical causes of retention are stricture, urethritis, the arrest of a calculus in the urethra, prostatic enlargement, and the pressure of the head in parturition. Remarkable retention of urine is sometimes observed in hysterical persons.

In infants this condition may occur from phimosis, inflammation of the prepuce, or highly concentrated or acid urine. These causes may act reflexly, by producing spasm of the sphincters, or mechanically. The passage of a renal calculus through the ureter may, by reflex irritation, give rise to frequent micturition on the one hand or to spasm of the sphincters and retention on the other.

(f) **Suppression of Urine—Anuria.**—This condition may be mechanical, renal or general, partial or complete.

1. Mechanical causes of complete anuria are renal calculi blocking both ureters simultaneously or the ureter when only one exists. The symptoms are those of uræmia. The condition is extremely rare. Life may be prolonged several days with complete anuria; in Polk's case in which a solitary kidney was removed, the patient lived eleven days. *Partial anuria—oliguria*—may be caused by the presence of an abdominal aneurism or tumor upon one or both ureters, or by a kink in the ureter in the case of an ectopic kidney, or by malignant disease of the wall of the bladder involving one or both urethral orifices. In any of these conditions hydronephrosis may occur.

2. Renal lesions leading to suppression of urine are acute congestion, acute nephritis; the acute exacerbations of chronic nephritis, pyelitis, abscess of the kidney, perinephric abscess, and hydro- and pyonephrosis. Among the rare causes of suppression is thrombosis of the inferior vena cava or of the renal vein.

3. General conditions accompanied by suppression of urine are extreme lowering of the blood-pressure such as occurs in profuse hemorrhage from any cause; collapse or shock from injuries, surgical operations; the perforation of hollow viscera, as in peptic ulcer, empyema of the gall-bladder, enteric fever, or rupture of the uterus; the stage of collapse in cholera Asiatica, cholera nostras, or yellow fever, the pernicious malarial fevers, and acute peritonitis. Operations upon the urinary tract—even so trifling a procedure as catheterization—may in elderly men be followed by urinary suppression.

This symptom also occurs in acute poisoning by phosphorus, lead, and turpentine, in acute yellow atrophy of the liver, and in sunstroke.

Anuria, more or less complete, and prolonged for days, is occasionally observed in hysterical girls. In rare cases there are symptoms of uræmia, but as a rule there are no associated symptoms other than those due to the hysteria. In such cases, in order to avoid deception, the patient must

be isolated and carefully and continuously watched, and the catheter used at unexpected and irregular periods. Anuria may result from reflex irritation and functional arrest in a normal kidney, the ureter of the opposite side being blocked by a calculus, or the opposite kidney having been removed by operation.

Hæmaturia.—When small amounts of blood are present the color of the urine is smoky. With larger quantities it is bright red or even dark brown and opaque like porter. Erythrocytes are present, usually crenated or as rounded shadowy disks. The hæmoglobin is soon dissolved, especially in ammoniacal urines and those of low specific gravity. Blood from the kidneys is intimately mixed with the urine, which is discolored both at the beginning and at the end of the act of micturition. Clots are often present and they may be in the form of casts of the pelvis or ureters. Blood from the bladder may not appear until toward the end of micturition or at its close. Upon washing out the bladder the water returns tinged if the source of the hemorrhage be in the bladder but clear if it be in the kidneys. The differential diagnosis of the source of the bleeding, however, is often attended with difficulty and can be made only by means of the cystoscope or a differentiator by which the urine from each ureter may be obtained separately.

Hæmaturia may be symptomatic of the following conditions:

1. The hemorrhagic varieties of the acute febrile infections, forms of purpura, hæmophilia, very severe cases of scurvy, and leukæmia. A special form of hæmaturia or hæmoglobinuria—black water fever—prevails in certain malarious districts.

2. Diseases of the Urinary Passages.—Sarcoma or tuberculosis of the kidney, calculus in the ureter, tumor, ulceration or calculus in the bladder, parasites of the bladder—Bilharzia hæmatobia, psorospermiasis—or rupture of veins in its wall may be the cause of hæmaturia. In rare instances this condition is due to disease of the prostate. The arrest of a calculus in the urethra or acute gonorrhœal urethritis is sometimes attended by the passing of blood. This symptom occurs in strangury and there are cases of persistent hæmaturia in which no adequate lesion has been found.

3. Traumatism.—Hæmaturia follows operations upon the kidney. Gun-shot wounds or stabs involving the kidney, laceration of the organ from blows upon the back, falls or crushing accidents cause profuse bleeding. Similar injuries involving the bladder or prostate, falls or kicks resulting in severe contusion of the perineum and laceration of the urethra are also followed by hemorrhage, and this symptom frequently follows the use of the catheter.

(g) **Hæmoglobinuria.**—The urine is discolored by hæmoglobin, chiefly methæmoglobin. Red corpuscles are absent or few in number. The urine is smoky or brownish-red, even black, and upon standing deposits a dense, dirty brown sediment made up of granular pigment, the detritus of blood-corpuscles, epithelium, and pigmented urates.

Three forms are recognized: the toxic, the paroxysmal, and hæmoglobinuria of the new-born.

1. Toxic Hæmoglobinuria.—This variety is encountered in poisoning by those agents which produce rapid destruction of the erythrocytes.

Important among these are potassium chlorate, urotropin, pyrogallic acid, carbolic acid, arseniureted hydrogen, carbon monoxide, naphthol, and muscarine. It is also produced by the transfusion of blood from one mammal into another, by exposure to intense cold and violent exertion, and occurs after extensive burns. In malarial subjects it may follow the administration of quinine—*black water fever.*

2. Paroxysmal Hæmoglobinuria. — An affection characterized by the occasional passage of urine colored by hæmoglobin. It occurs in adults and is more common in males than in females. The paroxysms are excited by cold and exertion and last from a few hours to a day or two. It is thought by some observers to have an essential relationship to Raynaud's disease; by others to malaria. Pain in the lumbar region is common. The attacks may be ushered in by chills followed by fever; more commonly the temperature is normal or slightly subnormal. They recur at irregular intervals for an indefinite time.

3. Epidemic Hæmoglobinuria of the New-born. — The disease develops about the fourth day and attacks a large proportion of the infants in the maternity institution where it appears. There is bloody urine with vomiting and purging, jaundice, hurried breathing, and cyanosis. It is rapidly fatal. Post-mortem examination reveals enlargement of the spleen with punctiform hemorrhages upon the surface and in the parenchyma of the viscera. This disease is to be differentiated from icterus neonatorum to which it bears only a superficial resemblance.

THE REPRODUCTIVE ORGANS.

In both men and women sexual neurasthenia, hypochondriasis, and perversion frequently occur. Ungratified desire, excessive venery, and unnatural sexual acts are more commonly the alleged than the actual causes of various nervous and mental diseases. The two latter are probably manifestations more often than causes of such forms of disease. Irregular manifestations may be on the one hand psychical, on the other physical; frequently they are both. The field is a large one and the extent to which it is to be investigated in individual cases may be left to the judgment of the clinician.

The history or the actual manifestations of venereal disease in a patient, or in an individual with whom the patient has had sexual relations, are often of great importance in the diagnosis of an otherwise obscure case. A gonorrhœal discharge may solve the problem of an obscure and intractable arthritis or indicate the nature of serious tubal or other pelvic disease, and explain an unlooked for ophthalmia in the new-born. Syphilitic lesions or the scar of a chancre in the husband may be the key to the solution of obscure nervous symptoms in the wife, or nutritional disorders and lesions of the organs of special sense in the child.

In the male, priapism, impotence, and spermatorrhœa occur as important manifestations of disease.

(a) **Priapism.**—This term is used to designate abnormally frequent and prolonged erection. The condition is not associated with *libido sexualis* but with distress and pain and constitutes a morbid symptom.

It is often manifest in a mild degree in young boys. Even at the age of one or two years it may be painful and distressing and often leads to *enuresis nocturna.* It may be due to phimosis and disappear after circumcision. In the adult it may result from inflammatory irritation of the urethral mucosa. It may follow the passing of a bougie and is very common in gonorrhœa and in the chronic inflammation of the prostatic portion of the urethra in those who have practised masturbation or indulged in sexual excesses or irregularities. The condition may be due to excessive stimulation of the centre in the lumbar cord. The latter form comes on during sleep. The patient awakes with intensely painful priapism unattended by the slightest libido sexualis. This presently subsides only to return when, under the influence of deep sleep, the inhibition of the special spinal centre is withdrawn. In severe cases sleep is seriously interrupted and the annoyance of the patient is increased by the discharge of a thin mucus from Cowper's glands and painful neuralgia in various parts of the body. This form of priapism is often accompanied by impotence.

Priapism may be the result of stone in the bladder, inflammation of the prostate, a perineal abscess, proctitis or periproctitis, inflamed hemorrhoids, or poisoning by cantharides. It is said to be symptomatic of certain forms of neurasthenia and hysteria. It is a common symptom in fractures of the spine, especially when the cervical portion is involved. It may occur in myelitis, spinal meningitis, and in lesions of the pons and cerebellum. It occurs in hydrophobia and tetanus and has frequently been observed in leukæmia.

(b) **Impotence—Impotentia Coeundi.**—This symptom may be mechanical, psychical, irritative, or paralytic.

1. Mechanical impotence arises from congenital or acquired deformities; loss of substance from ulceration, gangrene, or operation; the presence of tumors, as hydrocele, enormous hernia, elephantiasis of the scrotum, and the like. To this list must be added hypertrophy of the organ, tumor of the glands, preputial or urethral calculi and defect, atrophy or destruction of the testicles. To this group of causes is to be added deviation of the erect penis from abnormally short frænum and various infiltrations and indurations in its tissues. A rare cause of impotence is deformity due to ossification of the fibrous tissue in the organ.

2. Psychical.—This form of impotence arises from apprehension, shame, or self-distrust. It may occur alike in those who have made too great experience and in those who have made none, and the fear of it frequently leads men about to marry to take medical advice. It is sometimes due to indifference, aversion, or dislike towards a particular person and in rare instances to constitutional lack of sexual feeling.

3. Irritative.—There is premature ejaculation or even ejaculation in the absence of sexual approach. This may occur in healthy individuals after long abstinence. It is very often due to local irritation, to lesions resulting from urethritis, or to excesses. The subjects are usually neurasthenic, the nervous condition being the cause in some cases, in others the effect of the sexual irregularity.

4. Paralytic.—Under this heading are to be grouped those forms of impotence caused by the loss of power to react to physiological stimuli on

the part of the sexual nerves or their centres. In the atonic cases anæmia and relaxation of the parts are present and the patients are neurasthenic. Sexual irregularities and excesses, immoderate indulgence in alcohol and tobacco are causes. Certain drugs, as opium and its derivatives, nitre, the salicylates and the bromides, taken in large doses or for long periods of time, lead to this form of impotence.

Diseases of the brain and spinal cord may be the cause of paralytic impotence. Tabes dorsalis and other affections, characterized by impaired function of the bladder or rectum or by local anæsthesia, are especially to be considered. This condition is also symptomatic of diabetes mellitus, obesity, and cachectic states.

(c) **Spermatorrhœa.** — This term is used to designate the pathological discharge of seminal fluid which takes place without erection or sexual sensation during the act of micturition or defecation. The emissions which occur at intervals of two or more weeks in continent young men during sleep, and which are accompanied by lascivious dreams, are physiological rather than pathological and are not to be considered under this heading. When, however, these emissions recur at short intervals, or every night, they become symptomatic of disease and the border-line between such nocturnal pollution and spermatorrhœa is no longer clearly defined. Gonorrhœa, onanism and sexual excesses are liable to be followed by spermatorrhœa. Constipation, nervous diarrhœa, fissure of the anus, seat-worms, and proctitis may act as accidental causes. The patients are neurasthenic and depressed, complain of headache, backache, and loss of energy, are much given to the reading of advertisements upon loss of manhood and are the easy prey of quacks. A large proportion of those who think they are victims of this disease do not have it, but suffer from chronic gonorrhœa, prostatorrhœa, urethrorrhœa, and forms of phosphaturia. The microscope is essential to the diagnosis, and it is necessary when spermatozoids are present to ascertain whether or not a sexual act has preceded the emission of the fluid in question. If not, and especially if spermatozoids are present upon repeated examination, the diagnosis becomes positive. These bodies are present in the urine, which may be acid, of high specific gravity, and contain oxalates, or alkaline with phosphates.

In the female pruritus vulvæ, leucorrhœa, and disorders of menstruation may be symptomatic of various local and general conditions.

(a) **Pruritus Vulvæ.**—This condition is a common result of inflammatory affections and displacements of the womb, ovarian disease, and affections of the urethra, bladder, and kidneys. It is, especially in children, a common manifestation of seat-worms and is very often the first symptom of the diabetic woman to attract her attention to her condition. This condition on the one hand frequently leads to masturbation; on the other is not rarely the result of it.

(b) **Leucorrhœa.**—Vaginal discharge is an important sign of many pelvic diseases. It is associated, very often in connection with pelvic inflammations of mild grade, with the anæmias, especially when intense, with conditions of debility and the later stages of chronic diseases when they occur in early life and in particular with pulmonary tuberculosis. In young children a purulent discharge indicates vulvitis or vaginitis, which

may be due to trauma, filth, ascarides, or gonorrhœa. In middle life an offensive sanguinolent discharge may be the earliest sign of carcinoma uteri.

(c) **Menstrual Derangements.**—The normal menstrual function may be deranged in various ways. It may be absent for a time or cease altogether—amenorrhœa; abnormally profuse—menorrhagia; or attended with much distress and pain—dysmenorrhœa. These derangements are due to local and to constitutional conditions.

1. Amenorrhœa.—Failure in the function may be a manifestation of arrested development of the ovaries and uterus. The interruption of menstruation may be physiological or pathological.

Physiological amenorrhœa is a characteristic sign of pregnancy and usually persists during lactation. There are important exceptions to both these rules. In very rare instances there is a slight menstrual discharge during the first two or three months of gestation and many women menstruate regularly during the period of nursing. Amenorrhœa occurs in extra-uterine fœtation.

Pathological amenorrhœa is observed in conditions of malnutrition, as in overworked school-girls, in those suffering from chlorosis, and in wasting diseases, as enteric fever, tuberculosis, diabetes, and exophthalmic goitre. It may be symptomatic of powerful depressing psychical states, as anxiety, worry, or grief, and of nervous affections, as hysteria, or of melancholia or other forms of insanity, and not infrequently occurs in young immigrants. It is common in morphinism and other drug habits and in cachectic states, whether due to chronic intoxication, as by mercury or lead, or to malaria, cancer, nephritis, leukæmia, or profound anæmia from any cause. The retention of the flow which takes place in cases of imperforate hymen, atresia vaginæ, and analogous conditions cannot be regarded as a form of amenorrhœa.

Delay in the establishment of menstruation is in some girls constitutional and often hereditary; its early cessation may in some instances be accounted for upon similar grounds. There are healthy women who cease to menstruate at thirty or thirty-five. Premature menopause may be due to atrophy of the ovaries following disease or their operative removal.

So-called vicarious menstruation, namely, the monthly discharge of blood from the nose, lungs, stomach, from hemorrhoids, ulcers or wounds, in the absence of the normal flow, is described. There is no physiological basis for such a phenomenon and it is probable that in the cases described the conditions causing amenorrhœa have also caused hemorrhages, the regular periodicity and duration of which have corresponded to the menstrual period less in fact than in fancy.

2. Menorrhagia.—Abnormally profuse menstruation may be symptomatic of disorders of the pelvic organs or of constitutional disease. It occurs in a great variety of local diseases but especially in chronic endometritis, submucous myomata, polypi, and uterine displacements. Menorrhagia is an occasional symptom in hæmophilia, scurvy, purpura hæmorrhagica, and leukæmia. When menstruation takes place in the course of the acute infectious diseases, for example influenza, enteric fever, or variola, it frequently amounts to menorrhagia. Other conditions in which this symptom is occasionally observed are intense jaundice, phosphorus poison-

ing, alcoholism, cirrhosis of the liver, and valvular disease of the heart. The administration of certain drugs, as ergot, gossypium, aloes, and the oil of savine, is sometimes followed by menorrhagia. Irregular menstruation, sometimes profuse, not infrequently precedes the menopause.

3. Dysmenorrhœa.—This term is used to designate collectively the symptom-complex in difficult menstruation of which pain is the chief element. The morbid conditions in which it occurs may be arranged under two headings, affections of the sexual system and general diseases.

Under the first heading are to be included those diseases in which there is an obstruction to the outflow of the menstrual fluid, as in contraction of the internal or external os uteri, congenital narrowing of the cervical canal or a narrowing acquired as the result of flexions of the uterus, the presence of tumors or cicatricial contractions following unwise treatment. This form is spoken of as mechanical dysmenorrhœa. Here also are to be considered the dysmenorrhœas caused by irritable or inflamed conditions of the mucosa secondary to chronic metritis, displacements, tumors and disease of the ovaries.

Under the second heading we include the dysmenorrhœa of neurotic persons—neuralgic or nervous dysmenorrhœa. This form is common alike in badly-nourished, anæmic, unmarried women and in women who have borne children. Very frequently no adequate lesions of the pelvic viscera can be discovered; more commonly trifling abnormalities such as cause insignificant symptoms in otherwise well-nourished and healthy women. The patients are neurasthenic and frequently hysterical. The symptoms vary greatly. In many cases they amount merely to an intensification of the ordinary discomforts which attend the periodical sickness; in others the patient may writhe with anguish or manifest the most intense reflex phenomena as nausea, vomiting, headache, or convulsions. Usually these symptoms subside upon the establishment of the flow; sometimes they continue with remissions and exacerbations throughout the whole period, and in some cases they cease entirely only to recur toward the close of the process.

Membranous dysmenorrhœa—decidua menstrualis—a form of dysmenorrhœa in which, with recurring menstruation, hollow membranous casts of the uterus are expelled with great pain. These casts consist of a thickened menstrual decidua. They vary from membranous fragments to complete triangular casts of the interior of the womb, showing the openings of the tubes and the internal os. They are usually expelled upon the second or third day, sometimes later. The pains are paroxysmal and very intense and cease immediately upon the expulsion of the membranes from the womb. This form of dysmenorrhœa is sometimes encountered in women suffering from chronic metritis or endometritis. It is very chronic, sometimes continuing throughout the entire menstrual life of the individual. There is complete relief during the intermenstrual periods. The condition is to be differentiated from early abortion and extra-uterine pregnancy.

4. Metrorrhagia.—An abnormal uterine hemorrhage is to be distinguished from an excessive menstrual discharge or menorrhagia, with which it is, however, very commonly associated. It may occur in diseases of the reproductive organs or in certain general affections. Metrorrhagia due to

local disease usually indicates disease of the uterus and mostly the presence of new growths, namely, carcinoma, sarcoma, or fibroid tumors. The bleeding in carcinoma at first takes the form of an increased menstrual flow usually more and more prolonged and frequently accompanied by a more or less abundant watery discharge. The bloody discharge after a time persists during the intermenstrual periods and becomes wholly atypical. The occurrence of bleeding in women who have passed the menopause is very suggestive and renders an examination per vaginam at once imperative. The metrorrhagia of sarcoma and in particular of sarcoma involving the uterine mucosa presents similar characters. Subserous fibromata do not bleed. Those situated in the substance of the uterus, if near the serous surface, bleed little or not at all. Submucous fibromata bleed more or less freely. Necrotic changes in uterine neoplasmata are attended by a foul-smelling discharge in which shreds of broken-down tissue are present. The atypical bleedings which attend inflammatory affections are less frequent and less profuse. Those which are caused by mucous polypi are often profuse and continuous.

Exceptionally metrorrhagia occurs in valvular disease of the heart, especially mitral stenosis, and is said to have been observed in cirrhosis of the liver. This symptom occurs infrequently in the acute infectious febrile diseases, as enteric fever, measles, scarlet fever, variola, cholera, and malaria, and in phosphorus poisoning and scurvy. In the last the blood loss is sometimes copious. Difficulties arise in the differential diagnosis of the cause of the bleeding when the patient suffering from the foregoing diseases has also local conditions in themselves capable of causing metrorrhagia or when, during the acute illness or shortly before its onset, an abortion or miscarriage has taken place.

XIII.

GENERAL SYMPTOMATIC DISORDERS OF THE NERVOUS SYSTEM.

PAIN.

Pain is a symptomatic sensory neurosis. The pain sense is to be distinguished from the tactile sense, the pressure sense, and the thermal sense. It is, however, so closely associated with the last two that a considerable degree of pressure, unusual heat, or intense cold is accompanied by pain. Pain is in the strictest sense a symptom. It is purely subjective, hence its value in diagnosis is to a large degree dependent upon the individual peculiarities of the sufferer, the nature of the primary lesion or disease, and concomitant phenomena, many of which are objective. Judged by these standards pain is a symptom of the most varied intensity, from a trifling discomfort without direct diagnostic significance to agony so extreme as to cause death. The pain sense is universally distributed throughout the body, the only structures in which it is wholly lacking being the hair and

nails. Variations in the pain sense in different localities, probably due to modifications in the sensory nerve supply, must be invoked in explanation of the different kinds of pain in the various viscera and other anatomical structures. Etiological factors of the most diverse kind have to do with pain in its relation to time, as shown in its onset, course, and decline.

Pain is dependent upon consciousness. In profound coma, as that of surgical anæsthesia, consciousness and pain are alike wholly abolished. When consciousness is less completely impaired there are objective manifestations of painful impressions, though the patient, upon recovering, may have no recollection of pain. Pain may be absent in shock. Individuals usually make no complaint of pain during the period of shock following gun-shot wounds or other severe traumatism. Under these circumstances pain comes on as shock subsides.

Etiology.—Pain is functional or organic. The temporary pain in overworked muscles is functional. The pain in pleurisy and gastric ulcer is organic. Pain occurs as a more or less prominent symptom under the following conditions:

1. Excessive or unduly prolonged physiological activity, either physical, as in muscular strain or fatigue, or psychical, as in the headache which follows undue intellectual effort. The pains of parturition are physiological.

2. Traumatism of all kinds.

3. Circulatory disturbances. (a) Passive congestion. An example of pain thus caused is to be found in thrombosis of the crural vein, formerly known as *phlegmasia alba dolens.* (b) Active hyperæmia, for example, the cutaneous pain of local irritants, as heat, cold, mustard and the like. Pain in the region of the spleen after running is an example of visceral pain due to this cause. (c) Anæmia. Examples of this form of pain are headache upon exertion and the neuralgias.

4. Inflammation. Pain is a prominent symptom in all forms of inflammation.

5. Toxæmia. The offending substance or substances in the blood may be the result of (a) infection, as in the acute specific fevers and malaria; (b) incomplete or perverted physiologicochemical processes or the defective elimination of waste, as in the headache of uræmia and diabetes and the pains of gout, rheumatism, and lithæmia; (c) the action of drugs or poisons. Pain due to this cause may be hyperæmic, as in the head pain produced by amyl nitrite and quinine; inflammatory, as in the later stages of narcotic poisoning; purely nervous, as an abstinence symptom in morphinism and the pains of the chloral habit and lead colic.

6. Changes in the arteries. Examples of pain due to this cause are found in syphilis, chronic alcoholism, chronic lead poisoning, migraine, and aneurism. To this general topic must also be referred the pain in intermittent claudication and angina pectoris.

7. All organic painful diseases, abscess, tumor, both benign and malignant, and various diseases of the viscera, whether the pain be due to changes in the organ itself or disturbance of adjacent structures by pressure or displacement.

8. Caries and other diseases of the bones.

9. Neuropathic conditions, for example, neurasthenia, hysteria, tabes, dysmenorrhœa, and tetanus.

10. Reflex irritation, as the supra-orbital pain in indigestion and the various localized head pains of eye-strain, pain in the external auditory meatus in dental irritation, and coccygodynia in uterine disease. Analogous are the pains in the knee which occur in hip-disease and painful sensations due to the irritation of the nerve stump referred to the hand or foot, as the case may be, in an amputated limb.

The cause of pain is very often simple. In many cases, however, it is complex, two or more of the foregoing factors being operative.

Mode of Expression of Pain.—Pain must be studied subjectively, as we experience it in our own person, and objectively, as manifested by the movements, attitudes, and verbal descriptions of the sufferer.

Subjectively we know that certain external impressions give rise to the sensation of pain and that this sensation is accompanied by movements of withdrawal from the object causing the pain, by particular attitudes of the body and contortions of the facial muscles. Under certain circumstances there are inarticulate sounds, cries or groans expressive of pain; these phenomena are varied according to the suddenness and the intensity of the pain and its character.

Objectively we recognize in these phenomena a manifestation of pain in others. The gestures that are characteristic of different varieties of pain have been described by W. H. Thomson. In pains due to inflammation the patient avoids touching the painful part, or approaches it very cautiously. Thus the hand passes over an inflamed joint with a hovering gesture. If the pain be deeper seated the gestures are indicative of its distribution and the character of the inflamed tissue. Thus the substernal pain of bronchitis as indicated by the whole hand laid upon the sternum and passed over the chest. In pleurisy the location of the pain is indicated by the tips of the straightened fingers, the natural gesture expressive of the stabbing or lancinating character of the pain. Precordial pain, if severe, is indicated by the tips of the bent fingers. The gestures by which abdominal pain is indicated are equally significant. In pains associated with lesions of the intestines the open hand is passed over the abdomen with a rotary movement. In the localized pain of appendicitis the open hand is held over the affected area with the fingers lightly flexed. In peritonitis the tips of the fingers are used but they touch the surface very gently and cautiously. Local pains resulting from visceral disease or colic are indicated by less guarded gestures; radiating pains by a repeated sweep of the hand in the same direction; distention pains and colic by a firm pressure upon the abdomen; neuralgic pains by repeated firm pressing movements of the hand in the direction of the involved nerve. The lightning pains of tabes are often indicated by a quick sweep of the tips of the fingers along the limb.

The shrinking of the whole body or of a member from an object capable of causing or increasing pain is a characteristic gesture; so also is the limping gait in painful conditions of a lower extremity. For diagnostic purposes it is important to bear in mind the fact that limping is frequently due to restricted movement not necessarily accompanied by pain. Very

characteristic are the attitudes in certain painful affections: retraction of the head in meningitis, the shallow breathing and flexion of the trunk toward the affected side in plastic pleurisy, the strong bending forward in colic, the rigid trunk and flexed thighs in peritonitis, the semiflexion and immobilization of inflamed joints.

Sudden immobility of the whole body is diagnostic of angina pectoris.

The facies of pain constitutes a most important objective manifestation, whether it be the contorted, dusky pale face of sudden agony or the drawn and pallid countenance of prolonged and repeated suffering. Severe pain, especially when paroxysmal, is frequently accompanied by dilatation of the pupils, rapid respiration, flushing or pallor, free sweating, increased arterial tension, and sensations of faintness. Inarticulate sounds and involuntary exclamations are familiar objective manifestations of sudden and intense pain.

Some of the objective manifestations of pain are involuntary and cannot be simulated; others may, with or without the conscious intention to deceive, be feigned or exaggerated. By the verbal description we gain information as to the location, character, intensity, and duration of pain, and the patient's opinion as to its cause. The accounts are much modified by the temperament, power of expression, and general experience of the sufferer.

Not only the ability to express the subjective sensation of pain varies greatly but also the susceptibility. There are on the one hand individuals in whom the pain sense is but slightly developed; on the other those in whom it is present to an abnormal and excessive degree.

There are racial differences in the susceptibility to pain and the mode of expressing painful sensations. The Latin races manifest a greater susceptibility to pain than the Anglo-Saxons. Oriental apathy is proverbial. On the other hand Hebrews appear to have a peculiar susceptibility to pain.

The individual susceptibility is much modified by temperament. Phlegmatic persons suffer less and show such sufferings as they experience much less forcibly than those of sanguine or nervous temperament. The neurotic individual suffers in proportion to the instability of his nervous organization. The pains of hypochondria and hysteria are probably of central origin. They are of irregular distribution, inconstant, and occur independently of the recognized causes of pain. They are probably none the less real. They diminish in intensity or disappear when the patient's attention is diverted from them and are aggravated by suggestion. The painful aura of epilepsy is also of central origin. Fright, expectation, and dread intensify painful impressions.

Somewhat analogous to the influence of temperament is that of the power of expression. The manifestations of pain are sometimes much less marked in the rude and uneducated than those in the higher walks of life. Apathy is a striking mental condition in hospital patients.

Experience is not less important. Habitual exposure to hardship benumbs the pain sense. On the other hand a life of refinement and luxury exalts it. Prolonged suffering or frequent recurrence of painful sensations augments the sensibility and each recurrence becomes less endurable. There is a popular phrase to the effect that the patient is *worn out with pain*

The manifestations of painful sensations are much influenced by circumstance and motive. Consciousness of pain is greatly diminished during intense religious or other excitement and upon the field of battle. When the excitement subsides pain asserts itself. The repression of the manifestations of pain by religious fanatics, the stoicism of captives under torture, and the fortitude with which the brave endure suffering set common experience at naught and emphasize the purely subjective nature of pain as a symptom. Not uncommonly patients understate their sufferings either from motives of pride or reserve or in order to avoid operation or treatment. On the other hand patients frequently appear to overstate their sufferings in order to secure sympathy or for other obvious motives. Women are more susceptible to pain than men and according to circumstances manifest it with greater intensity or endure it with greater fortitude.

The patient's description of his sufferings, the character of the concomitant phenomena, and the presence of an obvious cause will enable the physician to form an estimate of the significance of pain. In young children, in certain forms of insanity, and under other circumstances in which patients are unable to describe their sensations the objective manifestations of pain are of diagnostic value in determining its seat and intensity. The physician must be on his guard in any particular case against under-estimating the importance of pain or being deceived by its unintentional or purposeful exaggeration.

Varieties of Pain.—Pain in the broadest sense may be considered as parenchymatous or neuralgic. In the former the terminal sensory filaments are irritated; in the latter the nerve-trunks, the sensory roots, or the sensory centres. Parenchymatous pain is as a rule less intense than neuralgic pain and the spontaneous remissions are less marked. In the former the pain in the whole affected region is increased by pressure, while in the latter, though in some cases the entire region is tender under pressure, the general rule is that the tenderness is localized to the course of the nerve-trunk, especially when it is superficial or overlies a bone or makes its exit through dense fasciæ—so-called *tender points*. An example of parenchymatous pain is that which occurs in visceral diseases and the diffuse headaches; examples of neuralgic pains are the various actual neuralgias which occur as primary affections in persons otherwise in fair health, in the cachectic and broken-down, and as secondary affections in gout, syphilis, and diabetes and the lightning pains of spinal disease, especially tabes. The pains originating from suggestion and autosuggestion and many of the forms of hysterical pain are of central origin and may be regarded as parenchymatous.

Pain has been described as acute, sharp, lancinating, dull, throbbing, grinding, shooting, burning, chilling, shivering, boring, creepy, griping or colicky, itching and formicating. These descriptive adjectives indicate not so much distinct variations in the quality of pain as the simultaneous recognition of other associated sensations; hence, the descriptions of pain are often complex or picturesque in proportion to the vividness of the patient's imagination and his powers of expression.

(a) Acute Pain—Sharp, Lancinating, or Stabbing.—These adjectives are employed to describe the pain which attends acute inflammations

of serous membranes, as pleurisy, pericarditis, and peritonitis; the pains of acute arthritis; acute neuralgias; the painful forms of neuritis; acute phlegmonous inflammation, and the pains of thoracic aneurism. The lightning pains of tabes belong to this group and are characterized by their suddenness, brief duration, and intensity. They are sometimes spoken of as shooting pains.

(b) Dull pain is symptomatic of inflammation of the mucous membranes and the viscera. It occurs also in chronic inflammations.

(c) Throbbing or pulsating pain is encountered in acute superficial phlegmonous inflammations. This is the pain of whitlow—paronychia.

(d) Grinding, burning, or gnawing are adjectives used to describe the pain which occurs in diseases of the bones and periosteum, in aneurism of the thoracic and abdominal aorta, in carcinoma of the viscera and of the breast. Pain of this kind sometimes occurs in lithæmic conditions and in the later stages of acute gout. The localized neuralgic pain in the head, known as clavus, and the persistent local pains which occur in some forms of tabes are described as boring.

(e) Aching pains are not unlike the preceding. They are usually persistent and intense and, when severe, throbbing. Aching is a term used to describe pains in the head, those resulting from dental caries and forms of neuritis and myalgia, especially lumbago—hence, *cephalalgia, odontalgia, rhachialgia.* The pains which occur in the initial period of acute infectious diseases, as, for example, variola, influenza, and dengue, and are referred to the bones and muscles, are of this character. They are frequently associated with painful sensations of chilling or shivering and, since they spread from one part to another, are often described as creeping.

(f) Burning pain occurs in the superficial cutaneous lesions caused by intense heat or the action of the sun's rays, and caustic applications. It is characteristic of certain forms of neuritis. Circumscribed neuralgias are frequently associated with the sensation of burning pain—*causalgia.*

(g) Itching pain occurs in irritable states of the mucous membranes, such as attend certain forms of conjunctivitis, some acute diseases of the upper air-passages, and hay fever and some forms of inflamed hemorrhoids. Formication is a term used to describe a sensation like that of ants or other insects crawling over the skin. It is occasionally painful.

(h) Griping or colicky pains are those which attend the overaction of the muscular walls of tubal structures. Flatulent or other distention of the stomach or intestines induces pain of this kind—popularly gripes or belly-ache. The pains upon overaction of the muscular wall of the intestines caused by indigestible food, cathartic drugs, irritant poisons, and certain infections, as those of cholera morbus and cholera Asiatica, are colicky. To this group belong also the intense paroxysmal pains which attend the passage of hepatic and renal calculi—*biliary colic; renal colic.* These pains are frequently spoken of as cramp, a term also applied to painful contraction of the skeletal muscles, as those of the calf, toes, fingers, the pains of tetanus and strychnine poisoning and those which occur in habitually over-used muscles in certain occupations—writer's cramp, piano-player's cramp.

(i) Tenesmus is the term used to describe the painful bearing-down or straining sensations which accompany expulsive efforts from the outlets of the pelvic organs under certain abnormal conditions, as urination when there is acute inflammation of the bladder, urethra, or prostate gland, or stricture; defecation in proctitis or inflamed piles or hydatid or other tumors compressing the rectum. The bearing-down pains of labor are tenesmic.

Pain is modified by physical and psychical influences. Among the former are pressure, mechanical irritation, movement, and rest.

Modifications by Physical Causes.—The pain which is caused by pressure and the increase of pain upon pressure are described as tenderness. This will be discussed later under a separate heading.

Mechanical irritation causes pain or aggravates it in inflammation and ulceration of mucous membranes, as in aphthous and other forms of stomatitis, angina tonsillaris, peptic ulcer and fissure of the anus, inflamed hemorrhoids, and in various lesions of the tegumentary structures. Even slight mechanical irritation of the normal mucous membrane of the orifices of the body causes pain, as the presence of a minute foreign body under the eyelid, the introduction of a probe into the nasal chambers, or the passing of an urethral bougie.

Movement aggravates the pain of wounds, fractures, and inflammations. The pain which attends acute inflammation of serous membranes is especially increased upon movement, as is to be observed upon full inspiration in pleurisy and upon flexion and extension of the thigh in peritonitis. Movement intensifies the pain of arthritis, hence the involuntary immobilization of the joints and the relief afforded by splints. Movement also greatly increases the pains of vertebral disease and neuritis. The pains of myalgia and of all acute inflammations involving the muscles are augmented by movement of the affected part. In many instances the pains of inflammatory conditions and of visceral disease are increased by the motion of the body in transportation.

Rest, upon the contrary, is commonly attended with remission of pain; functional rest, by its temporary disappearance, as in myalgia, the headache of eye-strain, headache from prolonged study, and the pain of gastric ulcer. The foregoing facts indicate the value of attitude, posture, and movement in determining the diagnostic significance of pain.

Cold and heat modify pain. Hot applications are usually soothing; cold applications only occasionally afford relief. The application of heat or cold to the spine may indicate the level of disease by local intensification of pain. Applications of heat or cold frequently enable the dentist to locate the offending tooth in diffuse pain involving the distribution of the dental branches of the fifth nerve.

Seasonal influences modify habitual tendencies to pain. The pains of chronic arthritis, gout, and neuralgia are worse in cold and damp weather, better when it is warm and dry. The influence of climate upon such chronic painful affections is similar; dry, equable, warm, inland climates being more favorable than those of the opposite characteristics.

Modifications by Psychical Causes.—Among the psychical influences which modify pain and its manifestations, intense emotion, excitement, pride, and fortitude have already been mentioned. Other influences of

more importance in diagnosis are diversion, preoccupation, expectant attention, suggestion, and autosuggestion. They may be active under certain circumstances and to some extent in almost any kind of pain; but they are agencies of especial importance in neurotic persons and in those suffering from hysteria, neurasthenia, and hypochondriasis. Not only are the pains for which there are no obvious physical causes augmented or diminished, or made to disappear or shift to other parts, by purely psychical influences, but even those which attend actual injury and manifest disease may be greatly modified for a brief period of time. In the hypnotic state pre-existing pain may be made to disappear and definite pain aroused with readiness. It is evident that persons of great determination may inhibit the manifestation of pain under the stress of powerful motives. There are also rare individuals who appear to be able to inhibit the sensation of pain.

Time.—Pain in relation to time may be occasional, constant, persistent, intermittent, recurrent, or paroxysmal. Pain that continues for any length of time shows marked remissions and exacerbations. The remissions are due to functional exhaustion of the pain sense.

Distribution.—Pain may be (a) diffuse or general, or (b) circumscribed or local.

Diffuse pain is symptomatic of the stage of onset in the majority of the acute febrile infections. It varies in intensity from a mere sense of malaise or general soreness, as in enteric fever, to the severe aching of influenza, dengue, or variola. It occurs also in angina tonsillaris, particularly the lacunar form, and in trichiniasis. Diffuse pains attend certain stages of some chronic diseases, as syphilis, lithæmia, and saturnine and mercurial intoxication. They are sometimes described as vague and are often shifting. They are probably peripheral in origin and due to the action upon the nervous system of toxic substances in the blood.

Circumscribed or local pain occurs as a symptom in the greatest variety of morbid conditions. It is in fact the most common and most important of the subjective manifestations of disease. Its value in diagnosis depends largely upon the ability of the physician to estimate the accuracy of the verbal description, the spontaneity of the accompanying objective phenomena, the anatomical relationships of the pain itself, the underlying pathological process, and the importance of alleged or manifest causes. Pain, and in particular local pain, may be a danger signal, a sign post, a gauge of the progress or extension of disease, a counter check to objective phenomena, or it may be to the unwary or ill-informed physician a delusion and a snare.

Feigned Pain.—The simulation of pain is common enough in malingering, neurasthenia, and hysteria. The motives of malingering are innumerable. In neurasthenia and hysteria they usually consist of a morbid craving for sympathy. The detection of simulated pain is in some cases attended with difficulties that are insurmountable. In malingering the simulation of pain is usually overdone. The distribution of the pain does not conform to known anatomical rules. Suggestion is of importance. The objective phenomena commonly associated with intense pain are wanting or incongruous.

To properly estimate the value of pain in an obscure case it is sometimes desirable to have the patient under the close observation of an experienced nurse or attendant or in a hospital for some days.

Significance of Pain.—In general terms local pain is symptomatic of disease of the part to which it is referred. Organic headache, angina tonsillaris, the pain in the side in pleurisy, in the abdomen in peritonitis, in the joints in arthritis, and various forms of pain due to traumatism, are examples of the relationship of local pain to local disease. As regards the anatomical structure involved pain may be tegumentary, muscular, osseous, visceral, or neural. Very commonly the pain is also limited to the region or organ affected. But there are numerous exceptions to these statements, and we find local pain frequently symptomatic of a pathological process in a distant part, or local disease causing pain in an extended area. The recognition of these facts is of cardinal importance in estimating the value of local pain in diagnosis.

Referred Pain.—A familiar example is the intense pain over the supra-orbital notch sometimes felt upon eating an ice. The organ affected is probably the stomach, the location of the pain being determined by the association of sensory nerves from that organ with the trifacial. Very curious instances of referred pain have been reported—a case in which rubbing the forearm caused pain in the chest; another in which rubbing or pinching a mole on the leg was attended by sharp pain in the chin.

Referred pains manifest themselves in:

1. Symmetrical Areas.—A case is reported by Mitchell in which a shell-wound of the right foot at once gave rise to burning pain in both feet. A shell-wound of the left thigh caused an immediate reference of pain to the same area on both sides, so that the patient supposed he was shot through both thighs. Again, an injury to the median and ulnar nerves was attended by pain in the opposite hand.

Allochiria is the name given to the phenomenon of pain or other sensation referred to a symmetrical area. It has been observed in tabes and in postdiphtheritic neuritis.

2. Functionally Associated Organs. — Pain in the mammæ is common in congestion of the pelvic organs and dysmenorrhœa; pain in the glans penis or testicle in renal colic; diffuse pain in the abdomen in the early stage of appendicitis.

3. Segmental Areas. — Visceral disease is frequently attended by pain and tenderness referred to areas corresponding to the nerve supply of a given spinal segment. The affected organs receive their sensory nerve-fibres from the same segment of the spinal cord from which arise the fibres of the sensory areas to which the pain is referred. In the words of Head: "As the sensory and localizing power of the surface of the body is enormously in excess of that of the surface of the viscera, an error of judgment occurs, the diffusion area being accepted by consciousness and the pain referred to the surface of the body instead of to the organ actually affected." Hence the pain in intestinal colic is referred to the whole abdomen; that of hepatic colic to the epigastric zone, and that of renal colic to the lumbar region. So also pain in the heart, lungs, liver, and stomach may be referred to areas innervated by the cranial nerves and nerves given off from the

cervical plexus, and the pain in disease of the pelvic organs is very commonly referred to the back. A striking example of this kind of pain-reference is seen in the pain and exquisite tenderness of the right hypochondrium sometimes encountered in diaphragmatic pleurisy.

4. Longitudinally Related Areas.—Pain arising in the course of a nerve may be referred to its terminal distribution. The pain in the stump, which appears to be in the amputated foot, is a familiar example. The lightning pains of tabes, the thigh pains in malignant disease of the rectum and in psoas abscess, and the pain around the umbilicus in vertebral caries are further illustrations. Sometimes the areas are not so directly related, as in the knee pain in hip-joint disease, the shoulder pain in disease of the liver, and the pain in the distribution of the ulnar nerve in angina pectoris.

Peripheral pain may be an early and suggestive symptom in organic disease of the brain and spinal cord. In meningitis the pains in the back and limbs may be very severe. The joints are frequently the seat of pain, which may be more or less constant or lancinating and paroxysmal.

Painful Crises.—Severe and prolonged attacks of pain, associated with functional disturbances and wholly independent of local organic disease, occur in some cases of locomotor ataxia and are known as the tabetic crises. They are (a) cardiac—intense precordial pain accompanied by a feeling of oppression and rapid and irregular pulse; (b) gastric, the most common—sudden severe pain in the epigastrium, with vomiting, rapid and irregular pulse, sometimes symptoms of collapse; there may be vomiting without pain or pain without vomiting; (c) laryngeal, which is comparatively rare—pain in the larynx with paroxysmal cough, inspiratory stridor and sensations of choking; (d) pharyngeal, also rare—painful acts of deglutition following one another at short intervals and lasting from some minutes to half an hour. Intestinal, rectal, urinary, and genital crises have also been described. Suddenness of onset, intensity, paroxysmal character, and abrupt termination are characteristic of these attacks. The absence of lesions in the affected viscera either during the attacks or in the intervals between them is of diagnostic importance. Errors in diagnosis are common.

Localization of Pain.

Superficial pains are mostly symptomatic of diseases of the underlying parts, but they may be referred.

Deep-seated pain attends inflammatory and ulcerative diseases of the viscera, mediastinal tumor, aortic aneurism, visceral cancer, and disease of the bones.

Pain may be unilateral or bilateral. The former usually attends morbid processes confined to the affected side; the latter those involving both sides or of central origin. This rule is far from being absolute. The pain caused by floating kidney is occasionally referred to the opposite side of the abdomen.

The more important local pains and their diagnostic significance are now to be considered.

Pain in the Head.—(a) **Headache** is a term used to designate pain referred to various regions of the head. It may be paroxysmal or con-

tinuous. The term *cephalalgia* was applied by the ancients to slight, limited, or transitory headaches; the term *cephalœa* to severe, deep-seated, and chronic pains in the head. Headache is in many cases a symptom of such importance and prominence that it overshadows all others and lends to the clinical picture its most characteristic feature, often at first sight its only obvious feature. Headache is a symptom very often significant when other phenomena are obscure. It thus acquires a high degree of diagnostic importance.

Organic and Functional Headaches.—Headaches due to lesions of the skull or intracranial disease are organic; those due to other causes are functional. In general terms headache is the manifestation of the irritation of sensory nerve-fibres caused by derangement of pressure or tension, inflammation, toxæmia, and reflex disturbances. It is probable that the meninges are chiefly concerned in the causation of headache. The substance of the brain in the lower animals does not respond to direct irritation by the manifestations of pain; and lesions of cerebral tissue not directly or indirectly involving the membranes may exist without causing headache. The meninges and especially the dura, on the other hand, are directly or indirectly implicated in those pathological processes which give rise to headache. The sensory nerve supply of the dura in the anterior three-fourths of its extent, that of the falx and probably that of the tentorium are derived from the trigeminus, while the dura mater of the posterior fossa is supplied with sensory fibres from the vagus. The trigeminus is the nerve of sensation to the scalp as far back as the vertex, while the posterior branches of the upper four cervical nerves supply the muscles and the skin of the back of the neck and the occiput. Sometimes headache is referred to the scalp; usually the pain is deep-seated and intracranial. In rare cases superficial headaches are essentially myalgic, the pathological condition involving the occipitofrontal, temporal, or sternomastoid muscles.

The following clinical considerations in regard to headache are important:

Distribution of Headache.—This pain is usually bilateral. It may be frontal, occipital, parietal, and temporal, vertical or diffuse. The area most commonly involved is frontal, next in order of frequency is diffuse headache, then follow in the order named vertical, occipital, and temporal. Headache often shifts from one part of the head to another and is not always confined to regions limited by anatomical boundaries.

Varieties of Headache.—Headache, according to the character of the pain, may be: 1. Pulsating or throbbing. Headache of this kind is symptomatic of circulatory disturbances; it is often diffuse. 2. Dull, heavy. This is the headache due to toxæmia; it is usually frontal, sometimes occipital. 3. Binding or constrictive; the sensation is often described as that of a tight band around the head; the focus of intensity is referred to the parietal regions. This is the headache of hysteria and neurasthenia. 4. Burning or sore; forms of headache diagnostic of anæmia, rheumatism, and lithæmia. 5. Boring or sharp. These headaches are symptomatic of hysteria and allied conditions; they are usually localized; one form is known as "clavus"—the sensation as if a nail were being driven into the head.

Headache may be transient or persistent. In the latter case there may be exacerbations and remissions, or occasional intermissions which may last for days or weeks. There may be slight, continuous headache with exacerbations of varying intensity. Headache of this kind is symptomatic of forms of reflex irritation, especially those arising from defects of accommodation. Persistency is characteristic of organic headaches such as occur in cerebral tumor or abscess or pachymeningitis, or those which result from excesses in tobacco or alcohol, syphilis, and uræmia. The headaches which occur after sunstroke are persistent, with brief and irregular periods of remission.

The headache following cerebral concussion is severe and protracted. It may be circumscribed and limited to a region corresponding to the seat of the injury or to the opposite side of the head. It is commonly associated with tenderness on light percussion. The headache following injury may be, on the other hand, diffuse. It is apt to be associated with vertigo, lassitude, and indisposition to mental effort.

Significance of Headache. — *Congestion.* — Headache may result from mechanical interference with the return of venous blood from the head. When produced by improper clothing it is slight and ceases upon removal of the cause; when due to venous obstruction from the pressure of tumors it is not usually severe. The headache caused by violent paroxysmal or frequently repeated cough is congestive.

Hyperæmia.—Headache is symptomatic of active cerebral hyperæmia such as follows excessive and prolonged mental effort, and results from the action of vasodilator drugs, as alcohol and the nitrites. This form of headache occurs in the initial stage of acute meningitis. The headache of cerebral hyperæmia, whether passive or active, is usually frontal or diffuse, often pulsating or throbbing.

Anæmia.—Headache occurs in the anæmia due to blood loss or other cause. It is a common symptom in chlorosis. Anæmic headache is commonly severe, usually frontal or diffuse, often attended by sensations of pressure and not rarely associated with vertigo and tinnitus aurium. The headache of anæmia is intensified by effort.

Inflammation.—Headache is characteristic of all forms of cerebral meningitis, both acute and chronic. It is usually at first localized, a fact of importance in the diagnosis of meningitis due to mastoid or ethmoid disease or disease or injury of the cranial bones. It, however, rapidly becomes diffuse. Meningeal headache is usually continuous with exacerbations of great severity. Headache in exceptional cases is absent in the early stages of gradually developing leptomeningitis. Sudden intense headache with painful rigidity of the muscles of the back of the neck and vomiting are early symptoms of epidemic cerebrospinal fever. Intense paroxysmal headache is a symptom of tuberculous meningitis. The headache of pachymeningitis is local at first, but later becomes generalized. Severe frontal headache, usually unilateral, is symptomatic of disease of the frontal sinuses.

Infection.—Headache is a common manifestation of infection. This headache is usually frontal, it may be occipital or general, is often neuralgic or superficial, soon becoming dull, deep-seated, and severe. Headache is

an important symptom of the stage of onset of the acute febrile infections. It is early and severe in typhus and associated with pain in the back and limbs. After a time it is followed by stupor. It is a constant symptom in the early stages of enteric fever but subsides spontaneously during the second week of the disease. It occurs at the onset of relapsing fever and persists until the crisis, when it commonly ceases altogether. The headache of influenza is diffuse with points of intensity in the region of the frontal sinuses and behind the eyeballs. It may be a troublesome sequel. Intense headache characterizes the period of invasion of smallpox and is usually accompanied by excruciating pains in the back and joints.

Headache occurs in early syphilis. The headaches of late syphilis are usually symptomatic of arterial changes, gummata, or meningitis. Headache is common in hereditary syphilis. Paroxysmal headache is symptomatic of malaria. It occurs in the hot stage of the paroxysm. It is persistent and intense in estivo-autumnal fever. Periodical headache may be the chief symptom in estivo-autumnal infection.

Toxæmia.—Some intractable headaches are symptomatic of chronic uræmia. They are frontal or temporal, intense, usually continuous, with irregular exacerbations. Headaches of the same general character occur in diabetes and in those suffering from the gouty diathesis. To this group we may refer the headaches of chronic lead poisoning, those occurring in gastro-hepatic derangements, and constipation. These headaches are intensified by alcoholic beverages and relieved by free purgation. Certain drugs cause headache. Full doses of quinine or the salicylates produce headache and tinnitus aurium. Opium causes distressing headache with floating sensations, nausea, and vomiting. All these symptoms are increased when the patient assumes the upright posture. Tense, vertiginous headache follows the administration of the nitrites in full doses. Headache is a significant symptom in chronic poisoning by lead, tobacco, alcohol, opium, and chloral. In the case of lead and of alcohol arterio-capillary sclerosis is coöperative. Opium and chloral headaches are often abstinence symptoms, occurring upon the withdrawal of the drug. Intense headache not unlike that of migraine frequently follows excesses in alcohol—the *acute alcoholism of debauch.*

Cerebral Abscess.—Headache is often very severe and persistent in cerebral abscess. It is apt to be associated with vertigo and pronounced mental dulness and irritation. Vomiting is common but not constant. Chronic brain abscess may present no other symptom than headache, vertigo, mental dulness, irritability, and physical depression. The pain is usually related to the region of the lesion; in ear disease it is referred to the parietal or the occipital region of the affected side. In abscess following disease of the nasal or ethmoid bones the pain is referred to the brow. In abscess from traumatism the focus of pain is located in the region of the injury.

Tumor.—Headache may be said to be a constant symptom of brain tumor. Its frequency and intensity vary according to the location of the new growth, the rapidity of its development, and in some degree to its character. Headache is more persistent and severe in cerebellar than in cerebral tumors; in those of the cerebral hemispheres than in those of the base and in those directly implicating the meninges. It is more prominent in tumors of rapid than in those of slow growth, without regard to the nature

of the pathological process. In general terms the nature of the tumor formation has no direct relation to the intensity of the headache, the exception to this rule being that gliomata are less painful than other forms of coarse intracranial new growths. Headache in brain tumor is sometimes dull and boring, sometimes lancinating, usually intense, often agonizing. It is commonly continuous with periods of intensification, but sometimes recurs with a regular periodicity suggestive of malaria. The fact that it is commonly worse at night has some diagnostic value. The focus of the headache in cerebral tumor may be in the region involved, in the brow or in the occiput, or the pain may be diffuse. The headache of brain tumor may be localized when of moderate degree, diffuse during periods of intensification. Light percussion with the finger-tips may elicit tenderness in a region corresponding to the tumor. The headache of pachymeningitis interna hæmorrhagica is usually at first referred to the vertex; later it becomes generalized.

Aneurism.—Headache, either continuous or paroxysmal, is the most common symptom of intracranial aneurism affecting the larger arteries at the base. The location of the headache has in general no definite relation to the position of the aneurism, though aneurisms of the basilar artery usually occasion occipital headache. Headache occurs in caries of the bones of the skull.

Neurotic States.—Headache is a very common symptom in neuropathic conditions. In neurasthenia it is frontal, occipital, or diffuse; it is apt to be continuous and is aggravated by mental application and physical effort. Its intensity is moderate and it is attended by sensations of pressure in the head, aching in the back of the neck, and spinal pains. Headache is very common in the interparoxysmal periods of hysteria. It is often referred to the vertex and may be severe and persistent. Headache is common in emotional and precocious children. It is frequently associated with brow pains, pains in the back of the neck, and intolerance of bright light. Headaches of this kind are allied to the headaches of hysteria. Headache frequently enters into the symptom-complex of the epileptic paroxysm. It may precede or follow the convulsive attack. In the latter case it is associated with drowsiness and hebetude. Headache is common in petit mal. In many cases of epilepsy it constitutes an important symptom in the interparoxysmal state.

Reflex Headache.—This form is often troublesome and persistent. This is sometimes the case when the direct symptoms of the local disease are slight or absent. Errors of refraction constitute a common cause of reflex headache. The pain is usually frontal, sometimes temporal, often occipital. The patient is frequently unaware of any defect in visual accommodation. The headache is usually aggravated by close or prolonged use of the eyes. Reflex headache may occur as a symptom in chronic nasal disease especially in affections of the accessory sinuses. It usually involves the temporal region or the vertex. It is associated with sensitiveness of the nasal wall of the orbit and hyperæsthetic areas on the mucous membrane of the middle turbinate bone. Headache is an important symptom of adenoid vegetations in the nasopharynx. It constitutes one of the forms included under such terms as "school headaches," "headaches of

the period of growth," and the like. Associated symptoms are mouth-breathing, mental dulness, and irritability. Carious teeth and exposure of the pulp not only cause toothache but occasionally also cause reflex headache. Disease of the auditory apparatus may be the unsuspected cause of persistent headache.

The headache of acute indigestion and gastro-intestinal catarrh is probably rather toxæmic than reflex.

The importance of headache as a manifestation of disease of the sexual organs is probably over-estimated; yet this symptom is very common in those of both sexes who suffer from actual disease of the reproductive apparatus or are the victims of psychical processes concerning such diseases. Very often these headaches are due rather to the attendant neuropathic condition than to reflex irritation.

Associated Symptoms.—Vertigo, nausea, vomiting, drowsiness, irritability, and hebetude are associated with headache with such frequency as to indicate a common causation. These symptoms are as a rule less constant and less severe in symptomatic than in organic headaches. Vertigo is a frequent attendant upon headache due to gastro-intestinal disorder; nausea and vomiting in acute toxæmia; somnolence in malaria, anæmia, and syphilis. In organic headaches the presence of this group of symptoms and their persistence are important and suggestive.

Headache is essentially a symptom and a careful examination and inquiry will reveal some general or local cause. Headache is to be differentiated from migraine—a paroxysmal neurosis.

Neuralgia differs from headache in the following points: The pain involves the trunk or branches of the nerve rather than its peripheral distribution. It is unilateral, localized, sharp, paroxysmal, and there are present the characteristic tender points of Valleix. Neuralgia affecting the first branch of the fifth nerve is sometimes attended with suffusion of the eye and œdema of the lids.

Functional and Organic Headaches.—The differential diagnosis between functional and organic headaches is of fundamental importance. Organic headache is commonly persistent, varying from time to time in intensity, sometimes undergoing violent exacerbations but rarely wholly absent. It often interferes with sleep. It is aggravated by mental or physical effort, by excitement, alcohol, and all conditions that increase intracranial hyperæmia. It yields less readily than functional headache to symptomatic treatment. It tends to progressively increase in severity and is in many cases ultimately replaced by the stupor, drowsiness or coma of the terminal stage of the disease. Associated symptoms, such as vomiting, vertigo, hebetude, and irritability, are of diagnostic importance, and double optic neuritis, convulsions, and localizing symptoms, as monospasm, cranial nerve paralysis, cerebellar titubation, forced movement, and hemianopsia, render the differential diagnosis between organic headaches and functional headaches in most cases an easy matter.

(b) **Pains in the Scalp.**—Myalgic pains have been already spoken of. They are usually frontal or occipital, increased by voluntary movements of the scalp and by pressure. Various affections of the skin are attended by itching and burning pains of moderate degree. Local dermatitis attended

with pain sometimes results from the injudicious application of hair washes containing excess of cantharides and sometimes from the action of pediculi; also from burns and scalds, from erysipelas, and from traumatism.

Diffuse wandering pains are often experienced in various parts of the scalp and are associated with tenderness of the skin. These pains are not confined to the ramification of nerve-trunks and cannot be strictly regarded as neuralgic, but they very frequently alternate with true neuralgia. A patient under my observation compared these pains to sheet lightning.

(*c*) **Pains in the Face.**—The most important is trigeminal or facial neuralgia, known also as *tic douloureux* and *prosopalgia*. Neuralgia of the fifth nerve is much more frequent than all other forms of neuralgia. The pain is spontaneous, paroxysmal, and unilateral. Neuralgic pains involving the ophthalmic division usually affect the supra-orbital branch and are known as brow ague or supra-orbital neuralgia. The pain radiates over the front of the head from the supra-orbital notch. It may be felt in the eyelid or the eyeball or at the side of the nose. Tender points are found at or above the supra-orbital notch, in the upper eyelid, and on the side of the nose.

The neuralgic pain may be referred to the eyeball itself. It may occur spontaneously or as the result of over-use of the eyes. It is attended with dimness of vision and lachrymation and may occur alone or in connection with other neuralgic pain in the region of the fifth.

Neuralgia of the superior maxillary division of the fifth nerve is referred to the region between the orbit and the mouth and the side of the nose. Areas of special intensity are upon the side of the nose, over the prominent part of the upper jaw and along the gum. Paroxysms are frequently induced by the use of the tooth-brush. When the inferior maxillary division is involved a focus of pain is frequently found just in front of the ear, or in the temple or opposite the point of emergence of the nerve from the foramen, or in the region of the parietal eminence, and sometimes a point at the side of the tongue.

In intense paroxysms of trifacial neuralgia the whole side of the face and brow is involved and there is reflex facial spasm—tic convulsif. Supra-orbital neuralgias are occasionally attended with vasomotor disturbance. In other instances a herpetic eruption occurs which is probably the manifestation of an actual neuritis. Intractable neuralgias of the fifth nerve occurring late in life are known as degenerative neuralgias and are associated with changes in the ganglion of Gasser.

Severe pains in the distribution of the fifth nerve accompany cancer of the tongue, lingual ulcer, and caries of the inferior maxilla. Caries of the teeth and exposure of the pulp may give rise to pain referred to the ear.

(*d*) **Pain in the Eye.**—Inflammatory diseases of the eye cause local pain. In acute conjunctivitis there is pain in the eyelids, accompanied by photophobia and lachrymation; in iritis pain in the eyeball and intense supra-orbital pain, which may radiate in the distribution of the ophthalmic division.

The pain of glaucoma involves the distribution of the trigeminus, having its focus of intensity in the eyeball or at the supra-orbital notch. In the acute cases it is agonizing and associated with depression, pallor, nausea, and vomiting. In the chronic form it may be subacute with par-

oxysms of great severity. As the disease begins with great frequency on one side there is a misleading resemblance to migraine. Increase of the intraocular tension, irregular or dilated pupil, with inactive iris, haziness, anæsthesia of the cornea, and various visual derangements are suggestive symptoms.

(e) **Pain in the Ear.**—The pain of acute middle-ear disease is intense, throbbing, increased by pressure in front of the tragus and by gentle traction of the ear. It is subject to exacerbations and remissions and often radiates to the side of the face. Upon spontaneous or surgical perforation of the tympanic membrane the distressing feeling of tension is followed by immediate relief. Tinnitus is a common accompaniment. Pain referred to the ear and the side of the head is a prominent symptom in mastoid disease. It is accompanied by tenderness upon pressure and localized œdema.

(f) **Pain Referred to the Mouth.**—Pain is a symptom of various forms of stomatitis. It is intense in aphthous stomatitis, a very trifling affection, and often wholly absent in cancrum oris, one of the gravest of diseases. In mucous patches and syphilitic ulceration pain is less conspicuous than in tuberculous ulceration. In carcinomata pain is a persistent and distressing symptom. In inflammatory and ulcerative conditions of the pharynx pain is a prominent symptom. It is excited by mechanical irritation and by the contraction of the pharyngeal muscles in deglutition. Pain is not a prominent symptom in epidemic parotitis and parotid bubo. It is excited, however, by the movements of the parts involved and accompanied by great tenderness upon pressure.

(g) **Sinus Pain.**—Pain is a prominent symptom in disease of the accessory sinuses of the nose, especially in those cases in which there is an obstruction to the outlet. Under these circumstances the pain may be extremely severe and accompanied by marked systemic disturbance, as fever, chilliness, headache, and malaise. The sinuses usually involved are the antrum of Highmore and the frontal sinuses. Free discharge of mucus or pus is usually followed by immediate relief, but there are chronic forms in which the pain is apt to be of a dull character and constant, with exacerbations in damp weather and after exposure to cold. The diagnosis of antrum disease may be confirmed by transillumination with an electric light.

Pain in the Body.—(a) **Pain in the Back—Backache; Rhachialgia.**—Pain may occur in any part of the back. It is more common in the lumbar and sacral regions than elsewhere. Pain in the back of the neck extending between the shoulder-blades is a common symptom in neurasthenia and hysteria.

Acute pain in the small of the back attends the period of onset of many of the infectious febrile diseases, especially influenza, dengue, variola, and cerebrospinal fever. It occurs also in angina tonsillaris and acute nephritis. Acute pain in the back, much aggravated upon movements of extension, as in rising after lacing one's shoes, is characteristic of lumbago. Unilateral, deep-seated lumbar pain of great severity is symptomatic of renal colic. Persistent pain of this kind attends renal calculus. This pain is aggravated by pressure over the kidney or sudden jarring of the body. Pain in the back is often present in floating kidney. Sacral pains are symptomatic of disease of the pelvic organs, especially uterine flexions and displacements, ovarian disease, disease of the colon and rectum, hemorrhoids, and urethral stric-

ture. Many of the pains in the lower part of the back are myalgic. Pains of this kind result from occasional or habitual overwork of the muscles or from traumatism in the form of contusion or strain, or finally from exposure to cold or damp, especially in lithæmic individuals. The pain of myalgia is increased by movement, cold, and pressure; it is relieved by rest in the recumbent posture and by hot applications.

Pain in the spine occurs in disease of the vertebræ. Traumatism, syphilis, tuberculosis, and caries from pressure, as in aneurism of the aorta, are common causes. The pain is local and corresponds to the segment of the column involved. It is increased by sudden pressure upon the head or shoulders, by jarring, by the application of heat, cold, and faradism, and is relieved by the recumbent posture and in some cases by suspension and a properly applied spinal jacket. Rigidity results from muscular spasm in the earlier stages and from ankylosis in the later. Various deformities occur. Pain is present in that form of arthritis deformans which involves the vertebræ—*spondylitis deformans—spondylose rhizomélique.* There are associated nerve-root symptoms, as anæsthesia and muscular atrophy.

Pain attends various diseases of the spinal meninges. It is local and often intense. There are symptoms of irritation in the course of the nerves. The more common causes are hemorrhage into the spinal membranes and meningitis. Muscular spasm and rigidity are present.

Diseases of the cord are more apt to cause radiating and referred pains than pain in the spine itself. The latter is felt in the lumbar region; the former, as nerve-root irritation, as girdle pains, and in the lightning pains of tabes.

(b) **Pain in the Side.**—1. The pain may be symptomatic of injury or inflammation of the skin, as abrasion, contusion, local dermatitis, or furunculosis. The last is common in the axillary region. In rare instances phlegmon or subcutaneous extravasations of blood may be the cause of severe pain. An inspection of the parts is necessary in all cases.

2. Myalgic pains are not uncommon. Pleurodynia affects the muscles on one side, usually the intercostals, sometimes the pectorals and the serratus magnus. It is more common on the left than on the right side. It is especially distressing since the muscles are in constant use in respiration. The movements are restricted on the affected side, but deep breathing, coughing, and forced lateral movements increase the pain. Tenderness is present often in a limited area. This affection may suggest intercostal neuralgia, from which it is to be distinguished by the more circumscribed area involved, the paroxysmal character of neuralgic pain, and the well-defined tender points. It is sometimes mistaken for pleurisy, but the absence of friction sounds is of diagnostic importance. Violent spasmodic flexion to one side is an occasional though rare manifestation of tetanus and is attended with great pain in the affected muscles. Side pains referable to the muscles are observed in some cases of trichiniasis.

3. Pains due to injury or disease of the bones may be referred to the side. Fracture of the ribs, periostitis, osteosarcoma, rickets, and some cases of osteitis deformans are to be considered. The diagnosis demands a careful examination of the area involved by inspection, palpation, auscultation, and in obscure cases by the Röntgen rays.

4. The pain of plastic pleurisy is referred to the inframammary region or the side. It is sharp or stabbing,—the stitch in the side,—increased on deep breathing and accompanied by friction sounds, in some cases friction fremitus and a dry cough. It may occur in previously healthy individuals, or be accompanied by slight fever and presently disappear; it is a secondary process in croupous pneumonia and develops during cancer, abscess, and gangrene when the surface of the lung is involved. It is a very common phenomenon in tuberculosis of the lungs and may be basic or apical.

5. Pain in the side may be due to visceral disease. Sudden tension of the spleen, as often occurs in boys after running, is accompanied by intense pain in the infra-axillary region of the left side. Heavy, dull, dragging pains are symptomatic of the splenic tumor of leukæmia and the malarial cachexia—*ague cake*. Renal colic is characterized by an extension of the pain from the lumbar region to the affected side and thence downward toward the groin. In biliary colic the pain frequently extends to the right side of the chest. A dull heavy pain in the side sometimes attends upward pressure upon the diaphragm such as occurs in an overloaded stomach or distended colon, rapidly developing ascites, or an enormous abdominal tumor. Pain, paroxysmal in character but not extremely intense, occurs in the early stage of some cases of pyelitis. Intense pain in the lumbar region, aggravated by pressure, is a symptom of perinephritic abscess. It is often referred to the hip-joint or the adjacent region or the inner aspect of the thigh. This pain is attended with fixation of the thigh, which is flexed to relax the psoas muscle, and the patient in walking stoops and throws his weight upon the sound side. The pain of hepatic abscess is usually referred to the back or shoulders; it may be most severe in the right hypochondrium. A duller, dragging pain is felt in the right side when the patient turns upon the left. The pain of angina pectoris is occasionally referred to the left side—fifth, sixth and seventh and even eighth and ninth dorsal areas.

6. Pain in the side is very often the manifestation of disease of the nerves themselves. Neuralgia may be the result of nutritional changes in the sensory nerve-roots, the course of the nerve, or its peripheral distribution. Intercostal neuralgia is very common. Women are more liable than men; adults far more liable than children. The left side is more frequently involved than the right. Neuropathic individuals especially suffer. Intercostal neuralgia is encountered in anæmic conditions, general malnutrition, gout, lead poisoning, malaria, cachexia, and chronic nephritis. The attack may follow exposure to cold. The pain is paroxysmal and burning or lancinating and there are characteristic *points douloureux*. Trophic or vasomotor phenomena may occur, as local œdema or erythema. The posterior branches of the lumbar plexus may be involved with pain in advance of the crest of the ilium extending along the inguinal canal and spermatic cord to the scrotum—irritable testis—or the labium majus. The pain of herpes zoster is intense and often persistent. It corresponds to the distribution of the eruption. The pain in caries of the vertebræ and aneurism of the descending aorta is referred to the distribution of the intercostal nerves.

(c) **Pain in the Chest and Abdomen.**—1. The skin may be the seat of pain in inflammatory diseases, burns, severe eruptions, and herpes zoster. Painful burns sometimes result from the unguarded use of sinapisms or hot-water bags. An inspection of the part is necessary.

2. Myalgia of the abdominal muscles may result from continuous cough. The epigastric pain in children suffering from measles is due to the cough. Muscular pain attends tetanus and some cases of strychnia poisoning. Trichiniasis is to be considered.

3. Periostitis and necrosis of the sternum, costal cartilages, and ribs cause pain in the anterior wall of the thorax. Resorption and ulceration from aneurism, malignant disease, syphilis, and enteric fever are common causes of painful lesions in these structures. Contusions, fractures, and dislocations cause pain.

4. Many visceral diseases cause pain in the chest and abdomen. It is an important sign of aneurism of the aorta. It is usually dull and persistent with frequent paroxysms in which it is sharp and lancinating. It is frequently severe when erosion of the chest wall or vertebræ is taking place. Anginose attacks may occur. Pain may be absent. Broadbent has spoken of aneurism of the ascending arch as the aneurism of physical signs; of the transverse arch as the aneurism of symptoms. Pain is the chief symptom in aneurism of the abdominal aorta. It is epigastric, paroxysmal, and radiates to the back and sides. Severe epigastric pain occurs in aneurism of the cœliac axis and the splenic artery. Pain may occur in mediastinal tumor, but it is much less common than in aneurism and does not have the radiating character so common in the latter affection. The pain of mediastinal abscess is substernal, throbbing, and usually associated with chilliness and profuse sweating. In plastic pericarditis pain may be absent. When present it is variable in intensity, usually mild, exceptionally severe, and frequently intensified by the pressure of the stethoscope. It is felt in the precordia or at the base of the ensiform cartilage. The pain of pericarditis with effusion is sharp and lancinating and intensified by pressure over the ensiform cartilage. It may be dull and dragging. Pain is not a symptom of endocarditis. It occurs in chronic valvular disease, especially aortic insufficiency, in which it is sometimes persistent and distressing. It is usually precordial, dull, and aching; sometimes sharp and radiating to the neck and down the left arm. Pain is much less common in aortic stenosis and is not a prominent symptom in mitral disease so long as compensation is maintained. Angina pectoris is characterized by paroxysmal, agonizing pain in the region of the heart, radiating into the neck and arms, especially into the ulnar distribution of the left arm, and often attended with the fear of impending death. Chest pain is common and severe in croupous pneumonia, pleurisy, and pulmonary abscess. It may occur in any part of the chest but is most common in the inframammary and mammary regions. In some cases of severe acute bronchitis substernal pain is a distressing symptom. Pain may be absent in diseases of the liver. It occurs in acute infectious cholecystitis and is paroxysmal and severe. It is referred to the region of the liver but may have its focus of intensity as low as the appendix or in the epigastrium. Intense paroxysmal pain is met with in cancer of the bile passages. Biliary colic is of common

occurrence in gall-stone disease. There is agonizing pain in the region of the gall-bladder, extending into the lower thoracic, epigastric, and upper abdominal zones and radiating to the right shoulder. Dull dragging pain with intense exacerbations associated with nausea or vomiting is encountered in so-called hypertrophic cirrhosis. Pain of a dull, aching character and radiating to the back and right shoulder occurs in hepatic abscess. Pain and uneasiness in the right hypochondrium are present in some cases of cancer of the liver. In pancreatic disease pain may be a prominent and suggestive symptom. It occurs in hemorrhage, acute pancreatitis, and abscess and is referred to the upper zone of the abdomen. It is intense and persistent with agonizing paroxysms. Painful colicky attacks with nausea and vomiting have been noted in pancreatic cysts and the passage of calculi has caused pancreatic colic. A dull pain under the sternum is present in inflammation and in spasm of the œsophagus. In cancer it may be persistent or only present upon attempts to swallow food. The pain of gastralgia is usually deeply seated; that of gastritis more superficial. Cardialgia is a term used to designate the uneasy and painful sensations in chronic gastritis, sometimes caused by the taking of food, sometimes present when the stomach is empty. Pain is a distinctive symptom of gastric ulcer. It is gnawing, burning, paroxysmal, induced by taking food, and referred to the epigastrium. It is also in some cases felt in the back at the level of the tenth dorsal vertebra. In peptic ulcer of the duodenum the pain is sometimes located in the right hypochondrium and may come on two or three hours after eating. Pain is an early symptom in cancer of the stomach and occurs at some period in almost all cases. It is usually epigastric but may be felt in the back or loins. It is usually burning or gnawing and rather continuous than paroxysmal, though it is aggravated after food. The gastric crises of tabes consist of intense paroxysmal pain in the stomach accompanied with vomiting and an excess of intensely acid gastric fluid. Intestinal diseases are accompanied by pain which may be colicky when the small intestine is involved and bearing-down when the colon is affected—the tormina and tenesmus of the older physicians. Abdominal pain of variable intensity occurs in acute and chronic catarrh, ileocolitis, proctitis, malignant disease of the intestines, obstruction, intussusception, ileus, and appendicitis. It is the first and most distinctive symptom of peritonitis. Inframammary pain upon the left side is a common symptom of fecal accumulations in the sigmoid flexure of the colon in women, and is relieved by free purgation. Renal colic may extend well into the abdomen upon the affected side. Pyelitis may cause suprapubic pain. Displaced kidney is usually a source of much discomfort; often of distressing pain. The paroxysmal pains known as *Dietl's crises* occur in this condition.

5. Lead colic, the referred pain of diaphragmatic pleurisy felt in the right hypochondrium, and the girdle sensations of disease of the spinal cord are abdominal pains of purely nervous origin. The last may be a mere sensation of a cord or belt around the waist or it may constitute an actual pain. It is usually upon the level of the umbilicus or higher but may be lower. The pain is sometimes much less marked upon one side than upon the other and may suggest a unilateral new growth or other form of one-sided abdominal disease.

Pains in the Extremities.—In general terms the diagnostic significance is the same for the arms and hands and for the legs and feet. The exceptions are mainly as follows: The pain of angina pectoris extends to the arms and especially to the left arm and involves the ulnar distribution. The pain in writer's spasm and other occupation neuroses involves the forearms and hands. It consists of irregular darting pains in the affected muscles and the usual pains attending the spasm upon effort. The pains of dactylitis, onychia, and paronychia involve the fingers. Gout occasionally affects the fingers, but usually the foot and especially the great toe. A group of painful affections are due to improper foot wear—ingrowing toe-nail, corns, bunions and metatarsalgia. The pains of flat-foot, varicose veins and varicose ulcer are to be considered in regard to the habitually erect posture. The especial liability of the knee and ankle to troublesome painful affections and the greater frequency of venous thrombosis in the lower extremity are due to postural conditions and the greater distance of the blood-vessels from the heart. Referred pains are common in the lower extremities. The pain in hip-joint disease and obturator hernia is often referred to the inner side of the knee; that of ovarian and uterine disease, fecal impaction, aneurism, and other abdominal tumors, to the inner surface of the corresponding thigh, and in rare instances that of acute disease of the prostate gland to the sole of the foot. Pains in the limbs associated with numbness and tingling have occasionally been observed in the prehemiplegic stage of cerebral hemorrhage. Pain in the toes, due to peripheral neuritis, is an occasional affection after enteric fever. The affection is not attended by the signs of inflammation and passes away in the course of some days. Painful muscular cramps in the post-dormitium usually involve the lower extremities and in particular the muscles of the calf of the leg. They occur in pregnancy, in gouty subjects, and in persons otherwise in good health. Similar painful cramps may attend violent exertion and exposure to cold, as in swimmers.

The painful affections common to the upper and lower extremities, aside from traumatism and the action of cold, as in frost-bite, involve the muscles, nerves, blood-vessels, articulations, and bones.

1. Pain is symptomatic of myalgia from unaccustomed or habitual overwork. It shows itself in athletes, dancers, horseback riders, pedestrians and soldiers after forced marches and is without diagnostic significance. Muscular pain occurs in various forms of myositis and especially in trichiniasis. General muscular pain is a symptom of rickets: It occurs in scurvy and is distinctive of infantile scorbutus, in which it is a prominent symptom upon both voluntary and passive movement of the legs. Painful cramp upon muscular effort—intermittent claudication—occurs in thrombosis and arteriosclerosis of the lower extremities.

2. Nervous pain is symptomatic of neuralgia—tender points; paroxysms, pressure aggravation; neuritis either intrinsic or from pressure; peripheral neuritis or neuromata. Diffuse pain below the knees is especially common in alcoholic neuritis. Sciatica, as well as brachial neuritis, which is the same thing in the upper extremity, is in some instances a neuralgia; in others a neuritis of the nerve or its plexus. It is almost always unilateral. Lightning pains occur in spinal disease, especially tabes. They are more

common in the legs than in the arms. They are sometimes localized. Bilateral neuralgic pains in the arms and legs are due to spinal cord disease as sclerosis, to general toxic conditions as lead or arsenic, to vertebral disease, or in the lower extremities to pressure upon the nerve-roots of the cauda equina.

3. Venous thrombosis—*milk-leg, phlegmasia alba dolens*—is often extremely painful. It occurs in lying-in women and as a sequel to enteric fever and other infectious diseases. A similar condition may occur in consequence of local pressure in the upper extremity. Pain, usually tingling or burning in character, occurs in the early stages of local gangrene, in ergotismus, diabetes, and Raynaud's disease.

4. The joints are especially liable to pain. Exquisite pain is experienced in the joint affection of rheumatic fever. The wrists, elbows, knees, and ankles are especially liable to involvement. Another exquisitely painful joint affection is gout. Arthritis deformans is attended by occasional outbreaks of pain, each of which results in an increase of the previously existing deformity of the joints. Many of the cases described under the term chronic rheumatism belong to this category. The pain in gonorrhœal arthritis is persistent and rebellious to treatment. That of ordinary synovitis is of moderate intensity. Pyæmic joints are usually exquisitely painful. Postfebrile arthritis closely resembles the joint affection of rheumatic fever. In spinal arthropathies—*Charcot's joints*—and in tuberculous joints pain is not always a conspicuous symptom.

5. All forms of periostitis are accompanied by pain. The subperiosteal hemorrhages of scurvy are attended with pain, which is also a common symptom in osteomyelitis and a group of cases of osteitis deformans.

TENDERNESS.

Tenderness is pain upon pressure. It usually but not invariably accompanies spontaneous pain. Intestinal colic and some forms of neuralgia are relieved by pressure. Tenderness may be present in the absence of spontaneous pain. This symptom is often of considerable diagnostic value, but being purely subjective it is liable to the uncertainties which modify the diagnostic significance of spontaneous pain. It is attended by objective manifestations, as wincing, flinching, exclamations of suffering, and the like. As in the case of spontaneous pain the allegations of the patient cannot always be depended upon. In certain cases tenderness may disappear when his attention is directed to other objects, or it may be present under the influence of suggestion or expectant attention, or finally it may be simulated in malingering.

A distinction is to be made between tenderness, which is pain upon pressure, and hyperæsthesia, which is an exaggeration of the sensibility of the skin. Tenderness is (a) superficial, namely, pain upon a very light touch; or (b) deep, that is, pain excited by pressure sufficiently firm to extend to underlying parts. Superficial tenderness is closely allied to hyperæsthesia and is usually coupled with a diminution of the power to recognize the nature of the agent by which the impression is caused—loss of tactile sensibility.

For practical purposes tenderness, like pain, may be best studied in relation to the parts in which it is localized and the anatomical structures involved.

The Head.—Tenderness of the scalp occurs during and after the attack in migraine, occipital neuralgia, and in hysterical conditions. Light pressure or the use of the comb or brush may excite pain. Local tenderness is present in traumatism, especially contusions, and subcutaneous effusions of blood. Diffuse tenderness may be elicited in myalgia of the occipito-frontalis muscle. Tenderness attends periostitis and caries of the skull. It is present also in gumma. Tenderness with or without local œdema is symptomatic of infection of the mastoid sinuses—*suppurative mastoiditis.* Localized pain is produced by tapping upon the skull in some cases of meningitis, tumor, and abscess of the brain—a symptom of minor importance.

The Face.—Tenderness immediately in front of the tragus is present in acute inflammation of the middle ear. Tenderness over the malar bone is symptomatic of abscess and malignant disease of the antrum of Highmore. The tender points in trifacial neuralgia are found at the emergence of the branches from the bony foramina and their penetration of fasciæ. There is occasionally also sympathetic tenderness at the occipital protuberance and over the upper cervical spines. Exquisite hyperæsthesia is encountered in some cases of neuralgia of the fifth nerve.

The Neck.—Localized tenderness is found in acute inflammatory conditions, as mumps, cellulitis—*angina Ludovici*—acute adenitis; in myalgia, the spastic rigidity of meningitis; in caries of the cervical vertebræ and in cervico-occipital and cervicobrachial neuralgia.

The Thorax.—Tenderness in the course of the spine occurs in meningitis, spondylitis, arthritis deformans involving the spine, periostitis, and in some cases of myelitis. It is a symptom of importance in neurasthenia, hysteria, and spinal irritation, and in lumbago. Pressure upon the tender points produces not only pain but also marked acceleration of the pulse—*Mannkopff's symptom.* Thoracic aneurism causing erosion of the vertebræ is a cause of tenderness in the dorsal or lumbar spine. Spinal tenderness may frequently be found in lumbar, subphrenic, and perinephric abscess, and has been observed in acute inflammation of the bronchial glands and in some cases of tumor of the mediastinum. In these conditions pain may be also called forth by sudden pressure upon the shoulders of the patient or by jarring the body, as by a misstep.

Tenderness attends periostitis and caries of the clavicles, sternum ribs, and cartilages. It may be present in these structures in the painful form of osteitis deformans, especially early in the course of the disease. It is found in abscess of the wall of the thorax, perforating empyema, and eroding aneurism. Tender points are present in intercostal neuralgia. Tenderness upon percussion is not uncommon in the infraclavicular regions in phthisis. The mammæ sometimes are tender at the menstrual period, in early pregnancy, in the condition known as irritable breast, which is a syndrome of hysteria, and in adenoma and malignant tumor. Tenderness is a symptom of pericarditis.

Abdominal tenderness is a very common symptom. It may be general, as in peritonitis, or local. The latter is usually present in a limited area, as the epigastric, hypochondriac, umbilical, hypogastric, or iliac regions; or the tenderness may be found in one of the quadrants of the abdomen. Sometimes the tenderness is distinctly focal, as in peptic ulcer, the McBurney point in appendicitis, the region of the gall-bladder, or pyosalpinx. In other cases it is diffused, with or without circumscribed areas of intensity.

Epigastric tenderness is a symptom in acute and some cases of chronic gastritis, pancreatitis, pericarditis, acute yellow atrophy of the liver, and disease of the gall-bladder and bile passages. It may be found in some cases of Addison's disease. One or more tender points are present in peptic ulcer. Tenderness in this region attends the myalgia of persistent cough and may be observed in hysteria and hypochondriasis.

Tenderness in the right hypochondrium is encountered in various diseases of the liver, as perihepatitis, congestion, acute hepatitis, abscess, cancer, acute yellow atrophy, and in diseases of the gall-bladder and bile-ducts, including cholelithiasis. In the last group of cases the tenderness may be confined to the region of the gall-bladder, or diffused over the hepatic area or even more widely; in the left hypochondrium in acute distention of the spleen, infarct, perisplenitis, pancreatitis, and fecal impaction; in either hypochondrium in diaphragmatic pleurisy; in both in influenza, relapsing fever, and the gastrohepatic form of estivo-autumnal malarial fever. Tenderness in the umbilical region may be elicited in peritonitis, enteritis, and enteric fever; in the right iliac region in enteric fever, appendicitis, renal calculus, fecal accumulations in the hepatic flexure of the colon, and in cancer; in the left iliac region in cancer of the sigmoid flexure and in some cases of membranous colitis; in either in pelvic inflammations and diseases of the tubes and ovaries; in both when any of these conditions are bilateral, and in hysteria. Hypogastric tenderness may be symptomatic of cystitis, inflammation of the pelvic organs, dysmenorrhœa, and hysteria.

The Extremities.—Cutaneous hyperæsthesia may be due to peripheral neuritis, especially the alcoholic form, neuritis involving a nerve-trunk in the course of which there are tenderness upon pressure and *points douloureux*, crural thrombosis, varicose veins; to periostitis, osteitis, osteosarcoma, arthritis, myalgia, myositis, rickets, scurvy, trichiniasis, or tetanus. Forms of arthritis especially characterized by pain and tenderness are encountered in rheumatic fever, the acute process in arthritis deformans, the gonorrhœal joint infection, gout, sprain, and tuberculosis. The hysterical knee is usually exquisitely painful upon pressure.

PARÆSTHESIA.

Paræsthesia is a condition of modification of normal sensibility. The phenomena are due to irritation of the sensory nerves in their course or distribution. They depend upon nutritive disturbances of the nervous system or the action of toxic or irritating substances in the blood. The itching of mild morphine intoxication is an example. Similar symptoms

occur in gout and lithæmia and are met with in neurasthenia and hysteria. Sensations of numbness, burning, stinging, itching, and formication are common. Coldness, weight, tenesmus, the girdle sensation, precordial constriction, tightness, throbbing, sinking, faintness, and debility also belong to this group of symptoms. The sensations are closely allied to pain and are often described as painful by the patients. They are wholly subjective and their value in diagnosis rests entirely upon the ability of the physician to estimate the patient's accuracy of expression and desire to communicate the truth. There is no objective method of testing his statements.

These perversions of sensibility are very common and in many instances constitute the principal if not indeed the only symptom of which patients complain. They are much more common in women than in men and in the well-to-do than in the poor. Common associated conditions are defective digestion, constipation, anæmia, and general malnutrition. Important etiological factors are overwork, worry, irregular or indifferent meals, the stress of life, too frequent child-bearing, prolonged lactation, and enteroptosis. Remarkable forms and combinations are described by women passing through the grand climacteric. Forms of general and local paræsthesia constitute important epiphenomena of many chronic morbid states.

Cerebral Paræsthesiæ.—Sensations of heat, fulness, pressure, and other abnormal sensations in the head—the so-called *cerebral paræsthesiæ*—occur in neurotic individuals and over-taxed brain workers. These abnormal sensations do not amount to actual pain, though they frequently alternate with it. They are often distressing and sometimes intense. They occur in adolescence and early adult life and are especially common in women about the time of the grand climacteric. They are, however, more common in men than in women and in those given to intellectual pursuits and of sedentary habits than among the laboring classes. They occur with great frequency in lithæmic and gouty individuals. These sensations are sometimes general, sometimes localized to the vertex, occiput, or forehead. They frequently persist for long periods of time, in some cases preserving the same character, in others varying. They are augmented by mental effort and by disagreeable emotions and intensified by introspection and attempts on the part of the patient to explain them to his physician. They are, on the other hand, minimized by diversion and suggestion.

Forms of Paræsthesia.—The paræsthesiæ may be best studied in respect of their character, since almost any of them may be referred to various parts of the body and all parts at different times. They are described in the most varied combinations, so that numbness and tingling, itching and formication, burning and stinging, coldness and tension, tightness and throbbing, and many others occur.

Numbness.—This is a common symptom in superficial injuries of the skin from cold or heat; the action of corrosive substances, as the mineral acids and carbolic acid; overdoses of certain drugs, as aconite and the bromides; injuries of nerves, neuritis, neuralgia in the stage of access and decline and in the remissions of pain; herpes zoster; peripheral neuritis from any cause and in the endemic form of the tropics, beriberi; hysteria, neurasthenia, tetany, tabes, the early stages of myelitis, and in cerebrospinal fever. Numbness may be a localizing symptom in coarse lesions of

the brain, as tumor or abscess. It may occur as a premonitory symptom in apoplexy and as the aura in epilepsy. The sensation is sometimes described as like that produced by a very mild faradic current. It is common and distressing in myxœdema and may be a troublesome symptom affecting the hands and feet in arthritis deformans. Numbness in the hands and feet constitutes the condition known as *acroparæsthesia*. Waking numbness occurs at or about the menopause. It involves the extremities and usually passes off as the day goes on and ceases when the patient becomes adjusted to the non-menstrual life. Numbness is sometimes associated with or alternates with burning and tingling.

Itching or Pruritus.—This form of paræsthesia is frequently associated with formication and is sometimes so severe as to be described as pain. It is also associated with burning, especially in inflammations of the skin such as occur in the exanthemata, as measles and scarlet fever. Itching of the scalp is a symptom of seborrhœa; of the lips and nose a symptom of herpes; of the eyelids a symptom of beginning conjunctivitis; of the anal region a symptom of hemorrhoids or ascarides; of the external genitalia in both sexes a symptom of saccharine diabetes, in the female of leucorrhœa and neurotic states. Itching of the whole surface is a troublesome symptom in aged persons, in certain subjects in winter, in others who are lithæmic or gouty, in hysteria, neurasthenia, and many organic diseases of the nervous system. It is a symptom of jaundice, and sometimes follows the administration of morphine, copaiba, and other drugs. Pruritus is an occasional symptom in chronic interstitial nephritis and chronic lead poisoning.

Coldness.—A common form of paræsthesia. It is often general, as in the chill, rigor, or shivering which marks the onset of an acute febrile infection as pneumonia, or constitutes the initial stage of ague. Under these circumstances the internal temperature is elevated. Sensations of coldness with a normal or subnormal temperature occur in myxœdema, profound asthenia from any cause, especially after hemorrhage, hysteria, neurasthenia, and in some forms of spinal cord disease, as tabes, lateral sclerosis, and syringomyelia. Coldness in the back is often experienced by persons who are suffering from pulmonary tuberculosis in the period of incipiency. Subjective sensations of coldness in the extremities are usually associated with actual low temperature and often with some degree of cyanosis. In other cases the sensation of coldness is referred to a circumscribed area, usually in the leg or thigh. The affected region feels as though in contact with a piece of cold metal or even a piece of ice. This symptom occurs in neuropathic persons usually in middle life and commonly in men. It has been observed in local injury to a nerve-trunk and in spinal diseases.

Heat.—Heat as a subjective sensation not dependent upon general or local elevation of temperature constitutes a common and distressing paræsthesia. When it amounts to pain it is known as *causalgia*. It is mostly localized. Flushing is accompanied by the sensation of heat. Flushes of heat are common in stout women at middle life, at or about the menopause, and in nervous persons with weak heart. Subjective sensations of heat are sometimes associated with the girdle sensation.

Weight.—This paræsthesia is likewise of common occurrence. It occurs in the chest as a symptom in severe acute bronchitis, asthma, pleural and pericardial effusion, and mediastinal tumor; also in great cardiac hypertrophy and dilatation and in valvular disease upon rupture of compensation. Substernal weight and oppression may be a symptom of acute indigestion or of an overloaded stomach and may precede hæmatemesis. The sensation may be referred to the epigastrium. Weight upon the chest occurs in hysteria and neurasthenia and constitutes the *incubus* in nightmare. It is symptomatic of enteroptosis and splanchnoptosis, ascites, and abdominal and pelvic tumors.

Tenesmus or Bearing Down.—This form is frequently so distressing as to amount to actual pain. The milder forms are encountered in over-distention of the bladder, straining at stool, and some varieties of dysmenorrhœa.

Precordial constriction or stenocardia accompanies the pain of angina pectoris. Similar sensations but much less intense are sometimes experienced in cardiac asthenia, myocarditis, fatty heart, pericarditis, and when the heart is displaced upward by large ascites or abdominal tympany. It belongs also to the wide group of sensations in hysteria and neurasthenia.

Throbbing.—Sensations of throbbing are felt in conditions characterized by vascular relaxation and nervous excitement. Among these are aortic regurgitation, anæmia, and paroxysmal states in hysteria and neurasthenia. Almost every part of the body may be the seat of these sensations. They affect the head in migraine and other intense headaches; the neck in front and laterally in cardiac hypertrophy and exophthalmic goitre; the precordia in palpitation; the epigastrium in the pulsating aorta of neurasthenia; and constitute a local symptom in phlegmon and aneurism. Throbbing is commonly associated with objective pulsations. Purely subjective sensations of fluttering are described by nervous women. They are often referred to the left inframammary region.

Faintness.—Faintness is a sensation attendant upon enfeeblement of the heart's action, whether due to physical or emotional causes. Hence it occurs in dilated heart, myocarditis, fatty heart, and all forms of anæmia, especially upon exertion; in hemorrhage, shock, collapse, and upon the too sudden withdrawal of fluid by the trocar or aspiration; and finally in fatigue, excessive heat, and intense pain. Faintness attends sudden depressing emotion and mental shock. Weakness and debility are attended by subjective sensations which are characteristic and important, since they are often danger signals in the absence of the objective phenomena of oncoming disease. Sudden sensations of weariness out of all proportion to effort—*fatigue symptoms*—are suggestive of neurasthenia.

XIV.

GENERAL SYMPTOMATIC DISORDERS OF THE NERVOUS SYSTEM (CONTINUED): VERTIGO; CONVULSIONS; TREMOR; FIBRILLARY TWITCHINGS.

VERTIGO.

Vertigo—literally a turning—is a symptomatic derangement of the nervous system governing the relationship of the body to external objects. It is of two kinds: *objective vertigo,* characterized by sensations of movement on the part of surrounding objects which are really at rest, and *subjective vertigo,* characterized by sensations of movement on the part of the individual himself. It is popularly known as *dizziness* or *giddiness.* This symptom attends organic intracranial disease, but is more common in peripheral or functional disturbance. Vertigo in which no underlying pathological condition is discoverable is known as *essential.* Vertigo is a common nervous symptom. It is often associated with headache. It may occur (1) in mild cerebral concussion; (2) circulatory disturbances, as cerebral anæmia and hyperæmia; (3) local nerve irritation, as mechanical irritation of the external auditory meatus, inflammation of the middle ear, or the application of electrical currents to the head. A special form of vertigo—*true auditory vertigo*—occurs in labyrinthine disease. (4) Vertigo is a common symptom in toxæmic conditions and is associated with headache in the period of onset of the acute infections, in many cases of acute and subacute gastrohepatic derangements, and in lithæmia. It is symptomatic of narcotic poisoning, especially that produced by alcohol, tobacco, opium, and the nitrites. It occurs also in aniline poisoning. (5) It is a common symptom in arteriosclerosis and (6) in valvular disease of the heart, especially aortic insufficiency, and in forms of degenerative myocarditis; (7) in neuropathic conditions, especially neurasthenia and epilepsy; (8) in reflex disturbances, such especially as arise from diseases of the visual apparatus or the stomach; (9) in organic disease of the brain, especially in tumor, cerebellar disease, in meningeal irritation and meningitis, and in brain syphilis. Finally, (10) vertigo results from mechanical causes, such as swinging, certain unusual postures, rapid rotary movements, and sea-sickness.

Vertigo varies in intensity from a trifling sensation of imperfect equilibrium—mere swimming of the head—to the most active and distressing sensations of rapid or irregular movement or whirling of the body or of surrounding objects.

The equilibrium of the body is maintained by muscular action. The nicely adjusted and constantly varying motor impulses necessary to equilibrium are determined in cerebral centres in response to sensory impressions which are as continuous as the motor impulses which respond to them. These sensory impulses are visual, aural, muscular, articular, cutaneous.

and visceral. Anything which suddenly deranges the continuous and systematized though unconscious sensory impulses from these structures causes a derangement of the nervous mechanism by which the body is maintained in its relation to external objects. This derangement manifests itself as vertigo. These sensory impressions are not felt in normal consciousness, but when they are interrupted or when the cortical processes by which they are converted into motor impulses are deranged consciousness in regard to them is perverted and vertigo results. For this reason vertigo implies a disturbance, not a loss of consciousness. In true vertigo consciousness is always retained.

Vertigo comes on suddenly and is commonly of short duration. In the objective form the floor or the bed on which the patient is lying appears to rise and sink and objects whirl around, usually in a definite direction. In subjective vertigo the patient himself appears to be whirling around or rising and sinking in space. These sensations are often accompanied by compensatory movements on the part of the patient which may result in a fall. Mental confusion, faintness, a sense of alarm, and nausea or vomiting are associated symptoms, which vary in intensity but are almost always present. When the vertigo is severe consciousness is impaired but not lost. The attacks continue to recur whilst the causal condition persists. The term *status vertiginosis* has been applied to persistent vertigo.

The following forms of vertigo demand separate consideration:

Aural Vertigo.—This symptom frequently arises from the pressure of accumulated cerumen in the external auditory canal or from the pressure of air against the tympanic membrane by a blow upon the ear, or the entrance of water in diving or surf bathing, or the too forcible use of the ear syringe. It may also occur, though it is not a common symptom, in cases of middle-ear disease or from the use of the Eustachian catheter. Vertigo occurring under the above circumstances is usually slight and transitory. Labyrinthine vertigo is the chief symptom in Ménière's disease.

Toxic Vertigo.—Vertigo which attends the onset of the acute infections is of no great importance and usually quickly passes away. That which occurs in gastrohepatic catarrh is commonly annoying on rising in the morning in persons of bilious temperament and sedentary lives, especially if they be addicted to the pleasures of the table. This symptom occurs also in acute indigestion and in lithæmic conditions. Vertigo is a very common drug symptom, which is, however, much influenced by habit and idiosyncrasy.

Cardiovascular Vertigo.—Vertigo is a symptom of cerebral anæmia. It occurs in sudden blood loss, cardiac asthenia, excitement, or sudden effort during digestion, upon sudden effort in myocarditis, valvular disease, and in particular aortic insufficiency. It occurs also in pernicious and other forms of anæmia, chlorosis, and leukæmia. Associated with tinnitus aurium it is very common in sclerotic changes in the branches of the cerebral arteries.

Neurotic Vertigo.—Vertigo sometimes occurs in epilepsy as an aura. It is not rare in petit mal. Vertigo is a common and distressing symptom in neurasthenia. The attacks are frequent but not commonly severe or prolonged. They are attended with nausea, though vomiting is not com-

mon. It is usually subjective and frequently reflex. Stumbling or paralyzing vertigo has been observed in exophthalmic goitre and as an endemic condition in certain cantons of Switzerland during the summer. There is a sudden loss of power in the legs with impairment of consciousness. Paroxysmal vertigo may occur in nervous individuals after excitement or fatigue. It is very distressing, occurring suddenly, accompanied with nausea and vomiting, and lasting sometimes for hours.

Reflex vertigo may be associated with the brow pains and other forms of headache which are symptomatic of errors in refraction or want of harmonious action in the ocular muscles.

Mechanical Vertigo.—This symptom attends sudden lowering of the head, whirling around, or swinging in individuals not accustomed to it, and is a very important part of the symptom-complex in sea-sickness and car-sickness. Mild persistent vertigo has been observed in elevator boys.

Vertigo of Intracranial Disease.—This is a very common symptom in diseases of the brain and its meninges. It is sometimes distressing but as a rule is of secondary importance to the headache, vomiting, and mental dulness with which it is commonly associated. It occurs at some time during the course of meningitis, cerebral abscess, thrombotic softening, tumor of the brain, and cerebellar disease. This form is of considerable importance in the diagnosis of cerebral syphilis.

Laryngeal vertigo, better called laryngeal epilepsy, usually manifests itself in neurotic adults. The paroxysm begins with tickling or irritation in the larynx, cough, partial loss of consciousness, and dyspnœa. Light tonic or clonic movements occur. The patients suffer from laryngitis, bronchitis, asthma, or pulmonary phthisis. The attacks recur as often as once a day or at longer intervals.

CONVULSIONS.

The term convulsion is used to designate a paroxysm of involuntary and more or less violent muscular contractions involving the voluntary muscles in general. The word spasm is frequently used in a more limited sense to indicate similar involuntary contractions of the muscles of particular parts of the body. We speak of general convulsions and local spasms. This distinction is, however, not always observed.

General Convulsions.—Convulsions are tonic and clonic. A tonic convulsion is an involuntary muscular contraction which is continuous and intense. It may be of brief duration, as in the beginning of the epileptic paroxysm; or prolonged, as in tetanus. A clonic convulsion is characterized by the rapid alternation of contraction and relaxation, as in the second stage of the epileptic paroxysm or in infantile eclampsia. The posture in tonic convulsions is forced and immovable; in clonic convulsions it is constantly changed. The arms and legs are alternately flexed and extended with more force than in ordinary movements, the body is violently tossed, and the muscles of the face contorted. The chief centre for convulsions is the cerebral cortex. Tonic and clonic convulsions may succeed each other, as in epilepsy, or may alternate, as in hysteria. Consciousness is often preserved in general convulsions of the tonic type, as strychnine poisoning and

tetanus, and usually lost in those of clonic type, as epilepsy and uræmia. A spasm may be confined to a muscle or a group of muscles; or it may extend to an entire limb or the whole of the body. A cramp is a painful tonic spasm affecting a single muscle or group of muscles, as the well-known cramp in the calves of the legs.

Etiology.—From the standpoint of etiology convulsions are symptomatic of (1) local irritation; (2) general cortical irritation, (a) from causes wholly unknown, (b) from the toxæmia of infection, (c) from various intoxications; (3) circulatory derangements; (4) inflammatory and degenerative processes involving the cerebral cortex; (5) convulsions are very often of reflex origin.

Convulsions are essentially paroxysmal. Even though the cause is persistent, the motor centres become exhausted and there are intermissions, as in uræmia. Again the paroxysms occur as storms, the cause exhausting itself in a single paroxysm or series of paroxysms and only again asserting itself after an interval more or less prolonged, as in ordinary epilepsy. In infancy, in the children of neurotic parents, and in neuropathic individuals convulsions frequently arise from the action of causes not capable of producing them at a later age or in normal individuals.

1. Local Irritation.—The motor areas may be directly involved in fracture, hemorrhage, cicatrix, or neoplasm, as in focal or Jacksonian epilepsy. The initial symptom may be a local spasm, involving the leg, arm, or face, the convulsion becoming generalized in the course of a few seconds or longer. Again the local irritation may be transmitted from a distance, as in tumor, abscess, or sclerosis.

2. The irritation may be general, (a) from causes wholly unknown, as in epilepsy. The paroxysm is frequently preceded by an aura; it begins with tonic spasm and loss of consciousness and is characterized by clonic convulsions. So characteristic is the latter stage, that general convulsions due to other causes are described as epileptiform or epileptoid. The paroxysm is followed by hebetude, drowsiness, or stupor, and may be replaced by a maniacal outbreak or other mental disturbance—*the psychical epileptic equivalent.* (b) The toxæmias of infection. General convulsions very commonly attend the onset of the infectious diseases in childhood. They occur at this period of life as the equivalent of the initial chill in the adult and are frequently seen at the onset of scarlet fever, measles, and pneumonia, and in other not well defined infections. They are frequent in rickets, which is the most important predisposing cause of infantile convulsions. They are early symptoms of that disease, and when convulsions occur in infancy without manifest cause rickets is to be considered. The convulsions of tetanus, strychnine poisoning, and hydrophobia are to be considered under this heading. (c) General convulsions occur in poisoning from aconite, prussic acid, and veratrum viride, and in chronic alcoholism and lead poisoning. Under this heading are to be included the convulsions of uræmia, puerperal eclampsia, and asphyxia.

3. Circulatory derangements are sometimes the cause of general convulsions which occur after profuse hemorrhages, and in the cerebral anæmia which immediately precedes dissolution. Violent general convulsions occasionally occur during the coma following sunstroke.

4. Inflammatory and degenerative processes involving the cerebral cortex give rise to general convulsions. Under this heading are to be considered the convulsions of cerebrospinal fever and other forms of meningitis, cerebral syphilis, general paresis, and pachymeningitis hæmorrhagica.

5. Convulsions are very often of reflex origin. Painful affections and excitation in the region of a sensory nerve may produce spasms. Examples of reflex convulsions are those following severe injuries, burns, those associated with renal or intestinal colic, a foreign body in the ear, intestinal strangulation, retention of urine, and phimosis. Dentition and intestinal worms are less common causes of convulsions than is generally supposed. Indigestion is a cause of convulsions in infants and older children. In whooping-cough convulsions are very common. They result from the asphyxia attendant upon a prolonged paroxysm, cerebral congestion, or hemorrhage resulting from such a paroxysm. In other cases they are to be attributed to the depressed condition of the nervous system caused by the disease itself. General convulsions have been attributed to enlargement of the thymus gland as a result of pressure either upon the pneumogastric or upon the trachea. They frequently occur in children in whom no cause can be discovered and may in such cases be regarded as idiopathic. In infants in whom an attack of convulsions has once occurred a predisposition seems to be established, so that similar attacks occur from indifferent or not recognizable causes. In infantile convulsions the attack is commonly preceded by restlessness, fretfulness, grinding of the teeth, and slight twitching. It may occur suddenly without premonitory symptoms. The initial cry so common in epilepsy is usually absent, nor are the successive stages so well defined. The spasm begins in the hands; the eyes are fixed and staring or strongly turned upward; the body rigid, and the face congested. The convulsion is at first tonic, so that respiration is suspended, but presently clonic convulsions set in, the eyes are moved from side to side, there are violent twitchings or alternate flexions and extensions of the limbs, contortion of the face, and retraction of the head. There is spastic flexion of the fingers, the thumb being against the palm—*clenched fingers*. These movements gradually cease and the child passes into a condition of stupor. There is usually slight elevation of temperature. Convulsions arising from indigestion and those which usher in an infectious disease are commonly single, but those due to rickets recur in series. In some instances one attack succeeds another until death ensues.

When the attack occurs in a healthy child, it may be due to acute indigestion or some form of peripheral irritation; when accompanied by high fever and vomiting it may be the forerunner of an acute infection, as scarlet fever, or of infantile hemiplegia; when it occurs in badly nourished or rickety children it is apt to be incomplete and to recur. The convulsions of infancy do not of necessity run on into epilepsy, but general convulsions occurring without apparent cause at irregular intervals in young children otherwise healthy are in a limited proportion of the cases epileptic from the beginning.

Uræmic convulsions may be preceded by headache and restlessness. Sometimes they come on without warning. The epileptic cry does not

occur, but in other respects the attack may resemble true epilepsy. The convulsions are often recurrent and prolonged, the seizures being separated by periods of coma or deep stupor. The temperature is usually subnormal; exceptionally it is elevated. The condition is recognized by the characters of the urine, the presence of œdema, the condition of the heart and arteries, a urinous odor, and the history of the case.

Puerperal convulsions present the same clinical picture as those which occur in ordinary nephritis.

Hysterical convulsions are to be distinguished from epilepsy by the emotional state which precedes the attack, the *globus hystericus*, the difficult respiration, the alternating laughter and tears. Sensations may be described which suggest the epileptic aura, as precordial, abdominal, or pelvic uneasiness or distress. The patient does not fall to the floor in instant and complete unconsciousness as in epilepsy, but gently or by preference upon a sofa or couch in such a way as to do herself no harm. The movements are irregular and clonic but usually much less violent than in epilepsy. The tongue is not bitten. The attack gradually subsides and the patient becomes conscious and emotional again. At the close of the attack a large amount of light-colored urine of low specific gravity is often voided. The more violent convulsions, manifestations of hystero-epilepsy, include grinding of the teeth, tonic spasm, opisthotonus, and other forced attitudes, clonic spasms, and more or less profound unconsciousness. The attack is more prolonged than in epilepsy and is followed by contortions and cataleptic poses and in some instances by attitudinizing suggestive of various passionate states.

In tetanus the earliest symptoms are slight stiffness of the neck and some embarrassment in mastication. These symptoms gradually increase until the condition of trismus or lockjaw develops. The spasm extends and involves the muscles of the body, causing the rigid attitudes known as opisthotonus, orthotonus, pleurotonus, and emprosthotonus. Respiration is interfered with by the muscular spasm and asphyxia may threaten from closure of the glottis. The convulsive paroxysms are excited by the slightest irritation and are of variable duration. Complete relaxation may not occur during the intervals. There is usually a history of trauma.

The resemblance of strychnine poisoning to tetanus is close. Trismus is absent as a rule and the relaxation between the convulsive paroxysms is complete. There is a history of the ingestion of the poison.

Tetany is characterized by the peculiar position of the hands and feet, the involvement of the extremities, less often the face and neck, and the presence of Trousseau's symptom—the reproduction of the paroxysm by compression of the affected part either in the direction of the principal nerve-trunks or over the blood-vessels; or of Chvostek's symptom—an increase in the mechanical irritability of the motor nerves, a slight tap over the nerve-trunk being sufficient to throw the muscles into active spasm. The history of the case is quite different from that of both tetanus and strychnine poisoning.

TREMOR.

Tremor is a rhythmical to-and-fro movement of limited range due to the alternate contraction and relaxation of opposing muscles. The movements are involuntary and differ from fibrillation in that they cause locomotion of the parts involved. It is due to nutritive alterations in the motor neurons both of the cortex and spinal cord. A distinction is made between *intention* or *volitional tremor*, which shows itself only upon intentional movements, and *passive tremor*, which occurs when the parts are at rest. The former is sometimes spoken of as paralytic; the latter as spastic tremor. In the examination the patient is to be observed at rest, in intentional movement, and in attitudes which require sustained tonic contraction of the muscles, as horizontal extension of the arms and hands, separation of the fingers, or protrusion of the tongue.

The following forms of tremor are of diagnostic importance:

1. The Intention Tremor of Multiple Sclerosis.—This form of tremor does not occur during rest, but shows itself upon intentional movement, usually at first slight, then progressively more rapid and with wider oscillations, so that the intended movement is greatly hindered. The movements in some cases are so great and so irregular as to suggest ataxia. The rate of the tremor in disseminated sclerosis is given by Peterson at 7.9 to 8.1 per second for the earlier stages and 4.6 to 6.3 for the later stages.

2. The tremor of paralysis agitans is distinctly slower. It continues during rest, becomes less marked upon movement, and upon determined impulse of the will may disappear for a brief period. The rate is from 3 to 6 per second. This form of tremor disappears during sleep. It usually first appears in the hands and is characterized by rhythmical movements of the index finger against the thumb which suggest pill rolling. The tremor of paralysis agitans very seldom affects the head.

3. Senile tremor is in its more moderate forms an intention tremor; in well developed forms a tremor of rest. The hands and arms are more commonly involved, but the head is often affected and the under jaw and lips. The rate is from 4 to 6 oscillations per second.

4. The tremor of exophthalmic goitre is best manifested in the hands when extended and the fingers separated. It sometimes affects the head. The rate is rapid—8 or more per second—and the excursion limited. Upon intentional movements the tremor is sometimes increased. This form of tremor is common in hysteria, in which, however, every form may be encountered. It is seen also in tuberculous meningitis, in lesions of the corpora quadrigemina, and rarely in disease of the cerebellum. Similar tremors occur in the acute febrile diseases. The tremor of enteric fever is an example. It occurs even in mild cases and is most noticeable in the tongue when it is protruded for examination. At first fine, it becomes coarser as the exhaustion increases. The lips are affected and in severe cases the hands. It is more marked in persons who are addicted to alcohol. Murchison regarded excessive tremor as one of the signs of deep ulceration of Peyer's patches.

5. The toxic tremors are usually fine. They are intensified upon intentional movement. The more common causes are alcohol, tobacco,

morphine, and mercury. In alcoholic tremor first the hands and then the lips are affected, and it is temporarily intensified upon the withdrawal of alcohol and diminished by its administration in increased doses.

6. Tremor due to miscellaneous causes, as intense emotion, excessive or prolonged muscular effort and extreme cold may occur in healthy persons and is without diagnostic importance. Popular phrases are *trembling with anger* or *fear* or *cold*, and *buck fever*, in the inexperienced hunter.

FIBRILLARY TWITCHING OR FIBRILLATION.

This is an involuntary, brief, sluggish contraction of groups of muscular fibres rather than of an entire muscle. It is manifested as a wave-like movement of feeble intensity just under the skin, not involving the muscle as a whole and producing no movement of the parts to which the muscle is attached. It may occur in a limited number of fibres at long intervals, or in successive groups of fibres in rapid succession. There are cases in which fibrillary contractions do not occur spontaneously but can be excited by tapping the skin overlying the muscle with the finger, and in those cases in which they occur infrequently they may be produced in the intervals by the same manœuvre. They often occur in healthy persons upon exposure of the surface of the body to cold air. Fibrillation is probably caused by a lesion which at once weakens and irritates the cell-body of the peripheral motor neuron in the anterior horn of the spinal cord (Lloyd). It is, therefore, symptomatic of progressive degenerative processes involving and gradually destroying the large ganglionic motor cells, and occurs in paretic and atrophic muscles when those changes are of nuclear origin. This phenomenon is especially seen in anterior poliomyelitis and in bulbar paralysis. It may be present in traumatic neuroses without paresis or atrophy.

Other morbid motor phenomena are discussed in the chapter upon the Examination of the Nervous System.

XV.

PSYCHICAL CONDITIONS, EMOTIONAL STATES, DERANGEMENTS OF CONSCIOUSNESS, INSOMNIA AND OTHER DISORDERS OF SLEEP.

PSYCHICAL CONDITIONS.

The consideration of abnormal mental phenomena comes properly within the scope of psychiatry. Mental derangements constitute at times, however, important symptoms in almost every department of internal medicine. The degree of intelligence, defects of memory, emotional states, and irritative and depressive derangements of consciousness are to be considered. Closely allied are insomnia and other disorders of sleep.

Intelligence.—The age, education, and social surroundings of the patient are to be considered. Derangements of intelligence are frequently

associated with impaired consciousness but may occur independently of it. Both vary greatly in degree. Slight intellectual defects often not recognized in the ordinary demeanor and conversation of the patient become apparent upon further knowledge or upon taking a careful clinical history. The lower grades, designated by such terms as dulness and stupidity, or an extreme degree, as idiocy and dementia, are immediately apparent in the facial expression and behavior of the individual. Not infrequently a falling off in intelligence is manifest in persons suffering from chronic incurable affections, as valvular disease of the heart, nephritis, tuberculosis, and cancer. Not only is the nutrition of the cerebral cortex impaired but the patient's range of thought becomes progressively more circumscribed. His interest in general affairs or the particular objects of his previous intellectual activity diminish in proportion as his interest in his symptoms and in the narrow life of the sick-room increase. Graver derangements amounting to absolute indifference, stupidity, or dementia are on the other hand observed in cerebral diseases, especially in tumors of the brain, progressive bulbar paralysis, multiple sclerosis, hemorrhage, thrombosis, embolism, and softening. In other cases stupidity or dementia may be the expression of a developmental anomaly of the brain, as in idiocy and cretinism. Of special interest is the derangement of intelligence which occurs in myxœdema, both that form which develops spontaneously and in the *cachexia strumipriva*. In this condition, which is closely allied to cretinism or indeed practically identical with it, the derangements of intelligence vary in degree from moderate apathy and indifference with slowness of thought associated with slowness of speech to a state bordering upon dementia. A transient abnormal exaltation in mental activity with a rapid flow of ideas and unusual facility of expression may attend hectic fever, the action of alcohol, and excitement due to other causes. A corresponding depression in mental activity is observed in the period of reaction. The patient who has been restless and talkative in the febrile period is depressed and silent during the sweating that attends the defervescence; the exhilaration of alcohol is followed by the depression of a physical if not a moral remorse; fervor of speech and energetic action give place to dulness and abstraction.

Mental dulness or confusion occurs independently of derangements of consciousness. Confusion of thought attends grave neurasthenia, cerebral tumor, arteriocapillary sclerosis, old age, and profound malnutrition. Slowness of apprehension and unreadiness in expression are usually characteristic of defective intelligence, but may indicate lesions of the nervous mechanism by which ideas are received and expressed, as in forms of aphasia.

Memory.—As age increases the memory becomes less accurate and retentive. In many old people in other respects in good health and intelligence failure of memory becomes pronounced. At earlier periods of life the integrity of the memory is dependent upon the same conditions of general good health as that of the intelligence. We find therefore very often impairment or loss of memory in local lesions of the brain such as result from hemorrhage or softening, which are not, however, necessarily associated with enfeeblement of intelligence. Weakness of memory is very often observed in the traumatic neuroses—a fact demanding attention since frequently this condition is attributed to malingering. Individuals

recovered from severe traumatic neurasthenia very often have but faint recollection of the events associated with and following the injury. Loss of memory occurs in epilepsy, bromidism, and chronic alcoholism, is common in insanity, and often complete in terminal dementia.

EMOTIONAL STATES.

Mental depression is very common in chronic and incurable diseases. It is sometimes purely symptomatic. More commonly it arises from pain and suffering or from apprehension in regard to the future. A high grade of depression characterizes hypochondriasis and melancholia. In deep jaundice, especially when chronic, depression is very common. Mental depression frequently attends diseases of the stomach, particularly those in which pain is prominent. Periods of depression occur during the menopause and in pronounced neurasthenia, hysteria, and in cerebral disease.

Emotional exaltation characterizes acute and chronic mania and is an important element in active delirium. During anæsthesia by chloroform, ether, and nitrous oxide the early derangement of consciousness is manifest by emotional excitement which is often intense. A similar condition is characteristic of alcoholic intoxication.

Instability of temper, irritability, and sensitiveness are very common in invalids. The testiness and outbursts of anger which occur in gout and the fretfulness and impatience of uterine disease are well known. Emotional instability and similar changes in disposition are frequently observed in pregnancy.

DERANGEMENTS OF CONSCIOUSNESS.

These may be irritative or depressive. Irritative derangements of consciousness vary in degree from mild emotional excitement to furious homicidal mania; in extent from perversion in a limited region of consciousness relating to a single idea or group of ideas to systematized delusions influencing the whole life of the patient. Irritative frequently alternate with depressive derangements of consciousness. Delusions, illusions, and hallucinations are irritative derangements of consciousness.

A delusion is an unfounded conviction or belief. It is very often absurd or ridiculous. Delusions that are persistent and based upon false ideas having a logical interdependence or sequence are known as organized. An expansive delusion is an insane belief in the individual's own greatness, power, or goodness. No evidence or demonstration is sufficient to convince a person of the falsity of his delusions. Examples of delusions that are common are the belief that individuals, almost always unknown, are conspiring to do the patient a serious harm, or that the patient is the Christ or Solomon or Queen Victoria.

An illusion is a false or misinterpreted sensory perception. The phenomena upon which it is based actually exist. A patient who mistakes the nurse for an officer of the law, or a bundle of rags for her baby, or ordinary household sounds for the voice of God is the subject of an illusion. Illusions are very often transient or momentary.

An hallucination is a sense perception not founded on objective reality. Hallucinations may relate to any of the senses. The patient who sees the figures of bystanders or hears whispering voices, or perceives a disagreeable odor or unpleasant taste, or feels upon his shoulder the pressure of a hand when none of these objects exist, suffers from an hallucination. Hallucinations are frequently persistent and distressing.

The Obsessions.—An obsession is an idea which dominates consciousness often to the exclusion of other thoughts and ideas. It comes unbidden and cannot be dismissed by any effort of the will. Nevertheless its nature and unreasonableness are usually fully understood by the subject.

Obsessions very commonly take the form of definite systematized fears relating to certain objects or conditions. These constitute the so-called *phobias*, as *kenophobia*, the dread of large or open spaces; *claustrophobia*, the fear of closed or narrow spaces; *agoraphobia* (ἀγορά, a market place), the fear of a crowd; *aichmophobia*, the fear of pointed instruments or weapons or the dread of being touched by anything; *metallophobia*, a terror of touching or handling a metallic object; *pyrhophobia*, a morbid dread of fire; and many other forms of persistent and dominating fear.

Doubt constitutes a common form of obsession. The mental uncertainty may be restricted to a single subject or set of subjects or embrace almost every affair of life from the simplest to the most important, recurring with intolerable insistence and refusing to be allayed by the demonstration of the actual conditions to which they relate.

Another group of obsessions consists in a morbid exaggeration of the activities of life. Those who are subject to them are possessed of a demon of unrest and are irresistibly impelled to be continually doing something or going somewhere, usually aimlessly and without fixed purpose, and always ready without adequate motive to change from one occupation to another or from the selected course to a different one.

Closely allied to this group of obsessions are those which consist in an irritable impulse to touch a spot or an object—*folie de toucher*—or to repeat certain movements, as returning to pass through a door two or three times before departing from it, and the like.

Fixed ideas are closely allied to obsessions and the terms are often used interchangeably. There are those, however, who distinguish between these two derangements of consciousness, namely, that an obsession is recognized by the patient as an abnormal train of ideas without basis in fact, while the subject of a fixed idea is convinced that it is based upon the conditions as they exist and perfectly normal under the circumstances.

The foregoing derangements of consciousness are permanent symptoms in insanity. They occur also in hysteria and neurasthenia and constitute important elements of delirium.

Delirium is an irritative derangement of consciousness characterized by restlessness, excitement, and incoherence. Periods of delirium may alternate with somnolence, stupor, or convulsions. There are two forms of delirium. In the *active* or *maniacal* the patient is wild and noisy. He sings, screams, shouts, tries to get out of bed, struggles with his attendants, and has to be restrained by force. His face is congested, his eyes bright,

his expression alert, excited, even fierce. The second form is *low* or *muttering*. The patient lies quiet, murmuring in a low tone, holding incoherent and often whispered conversation with imaginary persons, or occupied with vague fancies and taking no notice of what goes on around him. If aroused he may give a rational but brief reply to questions, quickly relapsing into his wandering dreams. This form of delirium is sometimes associated with restlessness. The patient moves in bed, may even try to get up, but is easily restrained. Between these two there are transitional forms attended with moderate restlessness and excitement. The patients are irritable, disturbed by trifles, and at times incoherent, though not boisterous.

Delirium develops very readily in persons of neurotic temperament and in early life. It may occur in any severe illness. It is especially common in fever and usually indicates a grave infection. In febrile diseases children are more liable to delirium than adults, just as they are more liable to high temperature. In general terms, there is no constant relation between particular diseases and forms of delirium. Active delirium is, however, frequently associated with the acute infectious fevers. The delirium of pneumonia is sometimes violent; in inflammatory diseases of the brain and in acute mania it is often furious. In fevers of ordinary intensity the delirium is of moderate type. It is muttering or wandering in the exhaustion of the low fevers and in the later stages of other acute diseases. Delirium may be present in uræmia and in poisoning by belladonna, cannabis indica, hyoscyamus, and opium, and a loud and boisterous delirium quite different from delirium tremens sometimes occurs in acute alcoholism. In enteric fever the headache usually ceases as delirium develops.

The onset of delirium may be abrupt or gradual. An outbreak of maniacal delirium has in rare instances been the first manifestation of an acute infectious disease, as enteric fever, typhus, or pneumonia. Cases have occurred in which under these circumstances individuals have been regarded as insane and placed in an asylum. Much more commonly delirium develops gradually, showing itself first in a certain confusion of thought upon awaking from sleep. In some cases delirium is absent during the day, coming on again and increasing as night approaches. Mild nocturnal delirium is sometimes seen during convalescence from pneumonia, enteric fever, and septic conditions.

The delirium of inanition occurs in wasting diseases and in starvation. It is not very rare in malignant disease of the œsophagus or stomach and occurs in cases characterized by intractable vomiting. The delirium of convalescence is probably a delirium of inanition. In this form of delirium the outbreak is sudden, usually in the early morning. There is feebleness of pulse and a relaxed and sweating skin with cold hands and feet. It is very often of brief duration, yielding in the course of some hours or a day or two to the proper administration of nourishment and stimulants. Maniacal delirium not uncommonly follows the epileptic paroxysm—*postepileptic mania*—or may develop as the psychical *equivalent* of the paroxysm.

The delirium of alcoholism—*delirium tremens*—is very characteristic. It is almost always associated with hallucinations which take the form of large numbers of small objects, as mice, bugs, serpents, which continually approach the patient and inspire abject and pitiable terror, or there are animals running around his bed or crawling upon the walls. The delirium

is busy. The patient is restless, his hands are constantly moving, he tries to get out of bed, but is usually tractable. Associated symptoms of diagnostic importance are tremor and sleeplessness, which are almost constantly present. A condition not unlike delirium tremens may develop in other drug habits. It has been observed after prolonged excesses in morphine, chloral, and paraldehyde.

Carphologia, literally a gathering of chaff, the picking at the bed-clothes, seen in the wandering delirium of grave fevers and profound exhaustion, is of unfavorable prognostic significance. The patients lie quiet, wholly oblivious of their surroundings, plucking with feeble hands at the bed-covering or grasping at imaginary objects in the air. These movements are dependent upon hallucinations.

Delirium is sometimes simulated by malingerers. Feigned delirium is to be recognized by the absence of other signs of illness, the want of the characteristic incoherence, and by the continuing sameness and limited range of the manifestations.

Depressive derangements of consciousness vary in degree from simple clouding of the ordinary consciousness to complete unconsciousness. They affect the entire field of consciousness. Loss of consciousness may be sudden or gradual, and is a symptom of great diagnostic importance.

Somnolence is the term used to describe the mildest degree. The individual is dull, drowsy, and indifferent, but retains an appreciation of his surroundings and can respond more or less intelligently when addressed. Naturally there is no sharp line of demarcation between this and the following progressive conditions.

Sopor, literally a sound or deep sleep, constitutes a more profound impairment of consciousness. The individual lies deeply drowsy and indifferent to his surroundings but can be aroused. To questions he replies in monosyllables and when aroused can move himself about and has a confused notion of his surroundings. Left to himself he sinks again into an abnormal drowsiness attended with muttering or snoring.

Stupor is partial or nearly complete unconsciousness. The patient cannot be aroused except with difficulty and then replies reluctantly and briefly to questions, relapsing at once into his previous condition. The expression of the face is dull and "stupid." He is still capable of swallowing.

Coma is complete loss of consciousness. The patient cannot be aroused from his insensibility. Perception and volition are wholly suspended. The face is expressionless, the respiration stertorous, the mouth open, the tongue dry. Swallowing is impossible, the sphincter ani is relaxed, there is urinary incontinence or retention. The breathing is frequently irregular. It may be irregularly interrupted or show the Cheyne-Stokes modification.

Coma vigil is a condition of profound unconsciousness attended by muttering delirium and carphologia. It is characteristic of this condition that the eyes are open and appear to follow the movements of the attendants. The prognosis is ominous.

Syncope—a swoon or fainting—is a sudden loss of consciousness, usually complete and transient, associated with pallor, coolness of the skin, and muscular prostration. It is a manifestation of acute anæmia of the brain resulting from failure of the heart's action. It may be caused in neurotic persons by sudden violent depressing emotions, as fear or horror.

or follow intense or prolonged muscular effort, or accompany hemorrhage or shock. It is important in all cases to make the differential diagnosis between suddenly on-coming coma and syncope.

Lethargy or trance is a condition of unconsciousness, more or less complete, which occurs in hysteria. It has been observed in rare instances after excessive mental application or exhausting disease and cases have been noted in which it has occurred in individuals otherwise apparently in good health. It differs from coma in resembling a deep and protracted sleep from which the patient in some instances may be partially aroused. The patient is usually pallid, the extremities relaxed, the eyelids closed, the eyes turned upward or to one side. The pupils vary in size but react to light. Respiration and circulation are greatly enfeebled. The temperature is subnormal. The attack varies in duration from some hours to several weeks. Cataleptic rigidity, or convulsions, may develop.

Catalepsy is a condition of impaired consciousness characterized by rigidity affecting the voluntary muscles. A limb or the body of the patient may be maintained continuously for some time in the same posture. The position of the limb may be passively changed with slight resistance, remaining in the posture in which it has been placed. This condition of increased muscular tonus has been termed "*waxy flexibility.*" The attack may last for a few minutes or for several hours. It is attended with complete anæsthesia of the skin and deeper tissues. The rhythm of the respiration is disturbed, the circulation feeble, the surface temperature depressed, and the reflexes impaired. The eyes are usually open; the pupils are dilated but react to light. The attitudes are sometimes bizarre and grotesque. As the attack passes away the power of muscular movement is fully regained. Catalepsy is a rare symptomatic disorder. It is encountered in hysteria, occurs in hypnotic states, and has been observed in cerebral disease, as tumor and meningitis, and in forms of insanity, as melancholia.

Coma may be easily recognized. Its diagnostic significance is often obscure. It occurs not only in cerebral disease but in the most varied constitutional conditions. It may be symptomatic of the following:

(a) Organic disease of the brain, either general, as acute encephalitis, cerebral syphilis, multiple sclerosis, and general paresis; focal, as intracranial hemorrhage, embolism, thrombosis or softening, tumor, abscess and thrombosis of the cerebral sinuses; disease of the meninges, as inflammation, the pressure from exudate, and subdural hemorrhage or tumor; or, finally, it may occur in the course of disease of the cranial bones. (b) Traumatism of the head, producing cerebral commotion or compression. (c) The pre-agonistic state in all diseases terminating fatally. (d) The fully developed febrile infectious diseases. Only exceptionally is coma under these circumstances complete. Early and complete coma occurs in the malignant forms. (e) Uræmia, in which it commonly alternates with convulsions. (f) The last stage of diabetes. (g) Forms of auto-intoxication analogous to diabetic coma in which β-oxybutyric acid or its derivatives are present in the blood. (h) Rare cases of septicæmia, pyæmia, carcinoma, and acute yellow atrophy of the liver. (i) Narcotic poisoning, especially by alcohol, morphine, chloral, and various poisonous gases, and the surgical anæsthesia produced by the administration of ether, chloroform, nitrous oxide, etc. (j) General convulsions, infantile eclampsia, and the epileptic par-

oxysm. (k) Drowning and asphyxia from other causes. (l) Sunstroke and similar conditions produced by exposure to excessive heat. (m) Hysteria.

The Associated Phenomena in Different Morbid States Characterized by Coma.—The diagnosis of the underlying condition is always important, often difficult, sometimes impossible. When the previous history can be obtained from the patient's friends the diagnosis is simplified. A child is seized with convulsions and vomiting and falls presently into coma. The fact that other children in the family suffer from scarlet fever justifies a provisional diagnosis of malignant scarlet fever. A man in middle life complains of headache and becomes comatose, with twitching of the face and general convulsions. Information to the effect that he has had polyuria with low specific gravity, small amounts of albumin, and casts, warrants a diagnosis of uræmia. A girl is found unconscious, pallid, with irregular respiration and occasional twitching of the face or extremities. It is of diagnostic importance to learn that she has been a highly nervous person who has just passed through some emotional stress and that the coma was preceded by tears and outbreaks of laughter—phenomena characteristic of hysteria.

The anamnesis is not always conclusive. It frequently happens that a patient suffering from chronic nephritis becomes comatose from cerebral hemorrhage and that a man who has been drinking falls into a coma not the manifestation of alcoholic intoxication but of fracture of the skull. The causal diagnosis of coma is attended with increased difficulty in ambulance cases and patients concerning whom no history can be obtained, seen for the first time in a comatose condition.

Cerebral Disease.—Coma occurring in the course of organic disease of the brain is usually preceded by such general symptoms as headache, vomiting, delirium, and somnolence, with varied local symptoms which depend upon the position and extent of the lesions and may be either irritative or paralytic.

Apoplexy—the Apoplectic Insult.—Premonitory symptoms are rare. Headache, ocular derangements, and paræsthesia of the extremities may occur but are not characteristic. The coma usually is sudden and complete and the condition is popularly spoken of as a "stroke." In other cases the coma develops gradually—*ingravescent apoplexy.*

Traumatism of the Head.—The history of an accident or injury is important. A careful examination should be made for contusion, laceration, or depression of the skull. If necessary the head should be shaved. Bleeding from one or both ears may occur in fracture of the base of the skull.

Infectious Diseases.—The antecedent conditions leading up to the coma are usually known. Coma under these circumstances may be a manifestation of the intensity of the primary infection or of some secondary process. Occasionally in grave enteric fever, very commonly in severe typhus and cerebrospinal fever, coma develops in the course of the disease and is not necessarily the sign of impending dissolution. Coma may occur under similar circumstances from an intercurrent nephritis with uræmia or from intercurrent cerebral hemorrhage, sinus thrombosis, or in the rheumatic fever attended with endocarditis from embolism. Coma occurs early in, or may even mark the onset of, the malignant forms of the infectious diseases, particularly scarlet, enteric, and cerebrospinal fever and the

pernicious forms of malarial infection. In the last there is the history of exposure in intensely malarial localities and of one or two recent well characterized paroxysms of ague.

Uræmia.—Uræmic coma may occur in acute or chronic nephritis.

Diabetes.—In saccharine diabetes coma very often attends the terminal condition, particularly in the young. Three forms of diabetic coma are recognized: (a) The patient after exertion is seized with sudden weakness, syncope, and somnolence which gradually deepens to coma and is followed in a few hours by death. (b) The early symptoms are due to pulmonary or gastric derangement or there may be some local affection, as pharyngitis, phlegmon, or carbuncle. The attack begins with nausea and vomiting. The breath has the peculiar sweetish, fruity odor of acetone. The onset of coma is gradual. Death occurs in the course of one to five days. (c) The patient without special previous symptoms is suddenly seized with violent headache and the sensation of profound illness and rapidly falls into deep and fatal coma. There are cases of diabetes in which the coma is due to some accidental cause, as uræmia, apoplexy, or meningitis.

Narcotic Poisoning.—In coma from opium and its derivatives the face is pallid, dusky, and slightly cyanotic, respirations and pulse slow, pupils equal and contracted, skin natural, and temperature normal.

In alcoholic coma the face is commonly flushed, sometimes pallid, occasionally cyanotic. The respirations are usually normal in depth and frequency. They are sometimes stertorous. The odor of the breath is characteristic, the pulse is at first frequent and full, later small and feeble. The pupils are equal, sometimes normal, more frequently dilated. The skin is usually cool and moist and the surface temperature lowered, especially under circumstances of exposure to cold or damp, when heat dissipation is favored. Convulsions are not common.

Poisonous Gases.—Coma develops under circumstances that make the diagnosis clear. It may result from the inhalation of carbon dioxide, as in disused wells, and carbon monoxide—illuminating gas, charcoal fire—a very common cause of accidental death and suicide. There can be no question about the coma of surgical anæsthesia. During this state various accidents may occur. Asphyxia has resulted from the falling back of the base of the tongue and from pulmonary œdema. Progressively deepening coma may terminate in death from failure of the cardiac or respiratory centres, and apoplexy may occur.

Convulsions.—Coma is very often preceded by general convulsions or alternates with them. It is frequently preceded by convulsions in the malignant forms of the infectious diseases, especially in children, and sometimes in dentition and the digestive disorders in young infants—*infantile eclampsia*. It follows the general convulsions of epilepsy. Coma and convulsions may alternate in cerebral syphilis, general paresis, and some forms of alcoholism. The alternation of coma and convulsions is characteristic of uræmia.

The coma of sunstroke is very often preceded by convulsions. The skin is excessively hot and dry, the face flushed, the respiration labored, the pulse frequent and full. The temperature ranges extremely high and may become that of hyperpyrexia. Upon venesection the blood is dark, thick, and flows slowly from the vein. The diagnosis is usually easy.

Epilepsy.—The diagnosis of postepileptic coma rests upon the history of the case, the convulsive seizure, the bitten tongue, the foam upon the lips, and the sudden profound loss of consciousness of no very long duration. The congestion of the face, stertorous breathing, urinary incontinence, and general muscular relaxation may suggest apoplexy, but the signs of hemiplegia are lacking.

Hysteria.—The unconsciousness of hysteria is commonly incomplete —lethargy or its intensification, trance. Its duration may extend over several days or weeks. True hysterical coma which is a further intensification of the foregoing is very rare. A condition of impaired consciousness suggestive of coma not infrequently enters into the symptom-complex in the grand attack of hysteria. It is usually preceded by the ordinary phenomena of the hysterical paroxysm: laughing, crying, convulsions, extravagant muscular movements, and the like.

For practical purposes the differential diagnosis between the coma resulting from opium, traumatism, alcohol, apoplexy, and uræmia is of imperative importance. Only in a correct diagnosis are to be found the indications for treatment. These are often immediate and urgent. Furthermore the diagnosis may have to do with questions of medico-legal interest. Definite diagnostic phenomena are to be systematically sought for.

Such points in the anamnesis as are available are to be obtained from the patient's friends or the bystanders. The immediate investigation demands an examination of the scalp and head for evidences of traumatism; of the eyes with reference to pupillary conditions and reactions, strabismus, and conjugate deviation; the face for blood extravasations, flushing, pallor, cyanosis, œdema, puffing of the cheeks, the presence of foam upon the lips, a bitten tongue, relaxation of the jaw, the odor of the breath, and the presence upon the lips or face of the stains of corrosive or other poisons. The character of the respiration is to be studied, the frequency, volume, and tension of the pulse, the sounds of the heart. The occurrence of fecal or urinary incontinence is to be noted, catheterization should be performed, and the urine examined for the presence of albumin, blood sugar, acetone, etc. The signs of hemiplegia are to be sought in the position of the head and eyes—conjugate deviation—in the greater relaxation of the mouth and cheek upon one side and the complete loss of muscular tonus in the arm and leg. The temperature must be taken in the axilla, and if found to be very low, in the rectum also. The signs of antecedent disease, general anasarca, great emaciation, various specific and other eruptions and scars, and the general condition of the viscera as determined by the methods of physical examination, such as the presence of effusions in the serous sacs, great enlargement of the liver or spleen, and the like, are to be in turn rapidly investigated. The stomach pump is often necessary for the diagnosis. If the conditions suggest the possibility of pernicious malarial fever an examination of the blood should be made for Laveran's bodies.

Not every case demands such comprehensive and elaborate investigation. Very often the condition underlying the coma is obvious at a glance. In other cases it is speedily revealed. Once in a while the true condition is not discovered without careful and prolonged study, and there are obscure cases which tax the resources of clinical medicine.

INSOMNIA AND OTHER DISORDERS OF SLEEP.

Insomnia—Abnormal Wakefulness.—These terms are used to designate a disturbance of the nervous system characterized by habitual incomplete sleep or periods of entire absence of normal sleep. Sleep varies with age, sex, and individual peculiarity. In very young babies sleep is practically continuous; a healthy child two years old passes half its time in slumber; the adult requires from seven to eight hours out of twenty-four; and aged persons not more than five or six hours. Women need more sleep than men. Workers in the open air require longer hours of sleep than those of sedentary habits. Insomnia may be functional or symptomatic. Functional insomnia occurs in neurotic individuals and over-taxed brain workers. Symptomatic insomnia is an important element in the symptom-complex of a great variety of morbid conditions. It occurs in painful diseases, as cancer, aneurism, and the intractable neuralgias. It is common in acromegaly. Insomnia is a very troublesome symptom in neurasthenia and various forms of insanity. It is an important element in acute delirium. Advanced disease of the heart is very often attended by sleeplessness due in part to cerebral anæmia, in part to the condition of the blood, but chiefly to the inability of the patient to lie down. As the condition progresses wakefulness gives way to somnolence and stupor. Tea and coffee have in many persons the power of inhibiting sleep. Complete insomnia is a conspicuous phenomenon in delirium tremens and alcoholic mania. Insomnia occurs with some degree of frequency also in the early stage of enteric fever, influenza, and croupous pneumonia. It is met with in cases of malaria and is a troublesome symptom in trichiniasis. It is not uncommon during the convalescence from acute disease. Insomnia may take the form of troubled and unrefreshing sleep of short duration or broken by intervals of distressing wakefulness, or sleep may be absent for days together. The patient may fall asleep upon going to bed but awakes in the course of two or three hours and lies absolutely awake or lightly dozes until morning. There is often great and irregular mental activity, especially in neurasthenia, and the cares, anxieties, and worries of the day are rehearsed with torturing iteration. Such insomnia is associated with restlessness, which is also present in the insomnia of insanity. Insomnia is rare in children but when present significant of profound disturbance of the nervous system. The sleeplessness of the aged is usually tranquil and unaccompanied by excitement or irritability.

Dreams usually have their starting-point in some sensory impression arising from local causes, as an uncomfortable posture, a sound which is perceived but which does not arouse, an over loaded stomach, a distended bladder or rectum, or a condition which interferes with the action of the heart and lungs. *Nightmare* is a frightful dream accompanied by sensations of oppressive weight upon the chest, intense fear, horror, or anxiety, and inability to move or cry out. The attack ends in a groan and the recovery of consciousness. It is mostly symptomatic of indigestion.

Night Terrors—Pavor Nocturnus.—This condition, which presents points of resemblance to nightmare and somnambulism, is a paroxysmal disturbance of sleep in young children. It differs from nightmare in the

gradual subsidence of the attack and the persistence of terror and distress after waking. It differs from somnambulism in the gradual waking, the less complete automatism, and the terror. The child starts up in bed screaming with fear and seeks protection, trembling and sobbing. The dream images are often indefinite, sometimes the creatures of imagination, suggested by the tales of the nursery. Night terrors occur commonly in neurotic and badly nourished children. They are sometimes symptomatic of eye-strain, the cutting of the second teeth, intestinal parasites, or indigestion.

Sleep drunkenness is a rare condition resembling maniacal delirium which appears upon waking from profound sleep. There are delusions of immediate danger to life or liberty. The sufferer fails to recognize his surroundings. He is excited, incoherent, and boisterous. The attack is usually of short duration.

Somnambulism—sleep-walking—is a disorder of sleep in which consciousness and volition are suspended but the activity of certain nerve-centres is exerted and coördinated movements are automatically performed. It occurs in adolescents and young adults of neurotic temperament and is more common in females. It is due to causes which ordinarily give rise to dreams, including indigestion, faulty attitude during sleep, intense excitement, or violent distressing emotion during the period preceding sleep. The attacks are frequently recurrent and may become habitual. They are of brief duration but may continue an hour or two, during which time difficult and complicated actions are performed, apparently with conscious intention. The eyes are closed or, if open, are staring and fixed. There is complete indifference to sound and the expression is blank and impassive. The patient on waking has no recollection of his wanderings.

Morbid Sleep.—Drowsiness may be symptomatic of cerebral malnutrition or toxæmia. It is common in aged persons with feeble heart and diseased blood-vessels, in the obese, and in malaria, anæmia, and diabetes. It is caused by the impure air of crowded assemblies. Cases have been reported in which prolonged deep sleep has ceased after the discharge of lumbricoid worms. Morbid sleep is a symptom by no means uncommon in organic cerebral disease, as syphilis, tumor, and arteriosclerosis. It is common in insanity, both in the prodromal period and the developed state.

Narcolepsy is abnormal deep sleep occurring in spells which may be of short duration or prolonged and continuous. The cause is unknown. In some instances the sleep has progressively advanced to deep and fatal coma.

Waking numbness—sleep palsy.—This is a form of paræsthesia occurring upon waking. There is a sensation of numbness and tingling. The distribution involves one or more extremities, usually the hands and arms. It is commonly of brief duration, disappearing in an hour or two. It resembles the forms of paræsthesia which occur about the grand climacteric.

Paroxysmal disturbances of the nervous system, both physiological and pathological, are common during sleep. Seminal emissions, the venereal orgasm, and urinary incontinence are accidents of sleep. Epileptic seizures—nocturnal epilepsy—are not uncommon, and the paroxysms of asthma and migraine frequently come on in sleep.

Focal Infection.

Much has been done in the matter of focal diseases and much remains to be done, and the absolute necessity of the harmonious association of clinicians and laboratory workers in research that is to be really productive has been more and more clearly recognized; but the work accomplished under the leadership of Billings and Rosenow constitutes an epoch-making contribution to the scientific basis of the art of medicine.

It is important that certain broad, general statements should be made at the outset. A focus of infection may be defined as a circumscribed surface or tissue invaded by pathogenic microörganisms. Such foci are primary or secondary. Primary foci may involve a mucous or cutaneous surface. They occur, however, commonly in the complex structures of the face and jaws, the upper air passages, the respiratory, gastro-intestinal and genito-urinary tracts, and the glandular and other organs severally connected with these. Secondary foci arise by way of the lymph-vessels in the form of infected lymph-nodes, or by way of the blood-stream in distant parts or organs. The lesions of such foci are frequently embolic and often widely disseminated.

Local inflammation results as an acute process with or without abscess formation, according to the nature of the infecting organisms, and is usually accompanied with more or less severe irregular fever, while insidiously developing visceral and cardiovascular diseases are among the manifestations of chronic focal infections which are often obscure and unsuspected. The endocardium and pericardium and the joints and periarticular tissues are particularly liable to secondary infection derived from distant limited foci of primary infection.

The most important recent contribution to the subject is to be found in the work of Rosenow, who demonstrated the transmutability of the members of the streptococcus-pneumococcus group in form, culture, characteristics and in general and special pathogenic virulence for animals and the fact that the property of transmutation is reversible among the members of this group. Scarcely less important is the demonstration of the fact that the bacteria of this group acquire elective tissue affinity in foci of infection, in culture media and in serial animal passage. To use the words of Rosenow, "The underlying conditions which tend most to call forth changes are, first favorable conditions for luxuriant growth and then unfavorable conditions—under stress or strain." And again, "It would seem, therefore, that focal infections are no longer to be looked upon merely as a place of entrance of bacteria but as a place where conditions are favorable for them to acquire the properties which give them a wide range of affinities for various structures.

The structures of the mouth and upper air passages are peculiarly exposed to infection. The great variety of pathogenic microörganisms present in the saliva and pharyngeal mucus constitute a universal danger of focal infection. The most trifling lesion of the mucous membrane serves as the point of entrance to deeper structures. To the *endameba buccalis*, present in the mouths of a great majority of persons, according to several

observers in as many as 90 per cent. of those examined, is attributed the most common initial injury to the edges of the gums. Similar lesions result from the maceration of food particles between the teeth and traumatism from toothpicks and other hard substances. Hence arise pyorrhœa dentalis and alveolar abscess. Enlarged faucial tonsils and hypertrophy of the adenoid and other lymphnoid tissues of the nasopharynx interfere with respiration and drainage and favor infection of those structures themselves, the middle ear and the accessory sinuses. In this manner foci of infection are established in a large proportion of children and many adults. Focal infections in the gastro-intestinal and genito-urinary tracts are less common.

The greater number of individuals thus affected do not suffer from acute or chronic diseases due to focal infection, being protected by the natural defenses of the body. Such persons are peculiarly liable, however, to the development of acute and chronic organic or systemic disease, the predisposing causes being profound emotional depression, the physical exhaustion resulting from exposure to cold, privation, insufficient food, prolonged illness, alcoholism, general anæsthesia and extreme age.

The most important acute disease of the organs of respiration is pneumonia, or to speak etiologically in view of the work of Cole and his associates, *the pneumonias*. Sternberg and Pasteur, by a curious coincidence, discovered in 1880 in human saliva a micrococcus which caused fatal septicæmia in rabbits and was designated the coccus of sputum septicæmia. Four years later Fraenkel demonstrated the fact that this bacterium is the most common germ in lobar pneumonia. Its causal relationship to pneumonia soon became generally recognized and it has been known from that time as the pneumococcus or diplococcus pneumoniæ. Notwithstanding the fact that more than three decades have elapsed since Fraenkel's discovery, no satisfactory comprehensive explanation of the occurrence of the infection under the widely varying conditions in which it takes place has yet been formulated. This explanation is supplied by the theory of focal-infection.

For this reason I place the pneumonias first among the diseases of the respiratory system caused by focal infection.

Members of the streptococcus-pneumococcus group have been shown to be present in the mouths of a large number of individuals apparently in good health, the proportion according to various observers being as high as 80 or 90 per cent. They vary greatly in virulence. In many persons they are apparently harmless denizens of the oro- and nasopharyngeal spaces; in others they are always virulent. In view of the transmutability demonstrated by Rosenow, changes in virulence and pathogenic selective affinity may be assumed as a working hypothesis. The virulence increases under conditions favorable to luxuriant growth. What these conditions may be is not yet fully known. They are doubtless the same depressing influences which by lowering the powers of the individual and weakening the natural defenses of the body favor systemic infection from any latent focus. Longcope and Fox found the saliva of the same person more virulent in cold weather.

The mucous membrane and secretions of the mouth and throat harboring virulent pneumococci constitute a focus of infection in the same sense

as the genital mucous membrane of the parturient woman infected by pyogenic bacteria. A focus of infection is not necessarily a small circumscribed lesion such as an alveolar abscess. It may consist of a great mass of infiltrated tissue, as, for example, a consolidated lung.

When systemic infection with virulent pneumococci takes place the primary localization is in the lungs; secondary invasions give rise to endo- and pericarditis, peritonitis, otitis, sinusitis, etc., and these affections are described as complications. The pneumococcus may be isolated from the blood. Meta-pneumonic empyema, the pneumococcic type, must also be regarded not as a *complication* but as an infective process, secondary to the pneumonia acting in turn as a focal infection. Thus an autoinfection occurs. This is in the great majority of instances the mode of origin of pneumococcus pneumonia—the *sporadic cases*. But there are occasional instances in which pneumonia is clearly acquired. The nurse after some days' attendance upon the patient has a chill, and forthwith develops pneumonia; and there are *house epidemics*, three or four cases, and less frequently more or less *extended local outbreaks*. Under these circumstances the infection is transmitted in the usual way. Pneumonia lends itself easily to transmission by droplet infection.

The conception of "focal infection" is based upon four fundamental facts:

(a) A circumscribed lesion or area of bacterial infection. Such a lesion is usually but not necessarily small; a pneumonic lung, a gangrenous uterus or a crushed limb may constitute the primary focus. Nor is it essentially a mass of infected tissue. The focal lesion in sporadic pneumonia and in diphtheria involves the mucous surface of the upper air passages; in erysipelas the inflamed mucous surface or skin and the neighboring or remote infective processes constitute so-called complications.

(b) The dissemination from the focus of bacteria by way of the circulating blood or lymph. If the focal bacteria are encapsulated or confined to a limited space under conditions which prevent their access to the body at large, infection does not occur—nor does it occur as long as they remain non-virulent or quiescent. These facts are of cardinal importance in the consideration of operative procedures.

(c) A systemic or local predisposition to the action of pathogenic organisms or toxins. There may be a natural or acquired immunity or the general defensive forces of the body may at times resist infection. Under these circumstances a focus of virulent bacteria may become a comparatively innocuous lesion.

(d) The absorption and systemic effects of bacterial toxins. Toxæmia having its source in an infected focus is an important factor in the causation of certain forms of general ill health and morbid conditions of various tissues and organs. It may exert its deleterious influence directly upon the nervous system, the heart and skeletal muscles, the kidneys and other organs or indirectly by lowering the power of resistance of special tissues or viscera and thus establishing a predisposition to bacterial invasion.

The pyogenic bacteria are the most common factors in focal infection, but all pathogenic bacteria may be present. Mixed infections are of frequent occurrence.

The effects of focal infection may be manifested in any tissue or structure of the body. The serous and synovial membranes are especially liable to infection. Sepsis, suppurative lesions, deep-seated abscess formations, endocarditis, especially the ulcerative form, arthritis, infected thrombosis are manifestations of focal infection.

The diagnosis involves the recognition of any particular morbid condition as due to focal infection and the location of the focus. The former is in general less difficult than the latter. At the time of this writing many sins are being committed against the teeth and the tonsils. The assertion that the removal of these organs as a diagnostic procedure is justifiable is not wholly true. Such operations are often without favorable result; sometimes followed by disaster from pulmonary embolism or hemorrhage or the establishment of postoperative infected foci in the locality in which such a lesion did not previously exist. Recurrent attacks of pneumonia, acute rheumatic fever, erysipelas and other paroxysmal diseases, such as gout, suggest the possibility of focal infection by bacteria of long periods of attenuation and brief outbreaks of virulence; insidiously developing visceral disease, in the absence of obvious cause, suggests the possibility of focal infection with resulting toxæmia, especially in view of the fact that occasionally marked improvement in health has followed the discovery and removal of a focus of infection. In all such cases the search should be systematically made. In this investigation the aid of the skilled specialist in many fields of medicine may have to be invoked. The condition of the nasopharynx, accessory sinuses and ears, of the jaws and teeth, of the joints and bones, of the genito-urinary and pelvic organs, of the rectum and prostate must be carefully investigated. Röntgenograms are of the greatest importance and should always be taken by an expert who devotes his time to that special work.

Schick's Test

The blood of a large proportion of normal individuals contains diphtheria antitoxin in quantities sufficient to constitute immunity against the infection. This test consists in the injection into the skin of an amount of diphtheria toxin equal to one-fiftieth of the minimum lethal dose for a guinea-pig weighing 250 grammes. If at the site of the injection there is no local inflammatory reaction in the course of twenty-four or forty-eight hours the individual very rarely acquires the disease. If such a reaction occurs immunity is absent and there is liability of the attack. It has been estimated that in the former case the blood contains one-thirtieth unit of diphtheria antitoxin or more per cubic centimetre while in the latter the content of the blood is less than this amount. The investigations of Schick, Park, Kolmer and others have shown that a very large percentage of the newborn and adults and from 50 to 60 per cent. of children between the first and fifteenth years possess relative immunity against diphtheria. The immunity of very young infants is probably, like that which they enjoy against other acute infections, transmitted from the mother, and the immunity of adult life may have been acquired by a mild or unrecognized attack at an earlier and susceptible age.

The outfit devised by Zingher of the Research Laboratory of the New

York City Department of Health, and consisting of a capillary tube containing the undiluted diphtheria toxin and a bottle holding sterile physiologic saline solution, is very convenient for the practitioner.[1]

Schick's test should be employed as a matter of routine in those who have been recently or are at the time exposed to the contagion and as a protection to the physicians, nurses, and other attendants in the diphtheria wards.

In the Philadelphia Hospital for Contagious Diseases persons yielding a positive reaction under these circumstances receive a series of three injections of toxin-antitoxin at intervals of a week. These injections consist each of a few drops of a standardized 90-per-cent. overneutralized solution of toxin and antitoxin, the amount of toxin remaining the same while the antitoxin is increased at each injection. The protection thus induced is of much longer duration than that resulting from the single immunizing doses formerly employed.

Acidosis

Notwithstanding the continuous formation of acid substances in the body and the great variations in the ingestion of foods containing acid and alkaline substances, the reaction of the blood remains nearly constant under normal and pathological states. This reaction is slightly alkaline. It is due to the presence of sodium salts and certain feeble acids, principally carbonic and phosphoric. When the acids are increased, they are neutralized by the sodium of the carbonates, and carbon dioxide is set free and eliminated by the lungs. In like manner the base of the phosphates neutralizes acids brought to them and the acid-phosphate remaining is eliminated in the urine. The carbonates and phosphates which thus stabilize the reaction of the blood have been called "buffer substances" (Henderson).

The term acidosis as it is now used in medicine is applied to a condition characterized by a general abnormal diminution in the blood of these buffer substances—namely, bases or substances which give rise to bases.

The designations "acidosis" and "acid intoxication" are unfortunate, since no marked change in the chemical reaction of the blood takes place and an actual acidity is incompatible with life.

Acidosis is always a secondary condition. The acid-base equilibrium is maintained by four principal processes—the excretion of CO_2 by the lungs; the function of the kidney in separating from the blood which is alkaline, urine which is acid; the capacity of the blood to dispose of considerable amounts of acid or alkali without appreciable changes in H-ion concentration and the formation of ammonia which assists in the neutralization of acid when the fixed bases are no longer available. When any one of these processes is deranged acidosis results.

Acidosis may be recognized by changes in the blood, modifications in the respiration, and alterations in the urine.

The changes in the blood furnish the most direct evidences of acidosis but require a difficult technic not available for ordinary clinical work.

Modifications of respiration are usually present: Air-hunger—*hyperpnœa*—is extremely common but not constant. Its occurrence always suggests

[1] Consult Jr. Amer. Med. Assn., lxv, pp. 329, 330.

acidosis. The changes in the blood stimulate the respiratory centre with the result that the pulmonary ventilation is increased and the CO_2 tension in the alveoli is diminished. The alveolar air may be collected by the Plesch-Levy method and examined by the Haldane apparatus for gas analysis. The normal tension varies between 39 and 45 mm. A reduction of CO_2 tension in the pulmonary alveoli may also occur in consequence of local changes in the respiratory centre.

The acetone odor of the breath is observed in cases attended with ketonuria.

The urine in acidosis yields information of importance when the kidneys are permeable. The ammonia may be increased or decreased according to the type of acidosis. In diabetes and cholera the ammonia coefficient usually rises; in certain types of nephritis it remains low. Acetone and the acetone bodies are not constant in acidosis, nor when present are they invariably the indication of its existence. They constitute the signs of a disturbance of carbohydrate metabolism and ordinarily increase in diabetic acidosis and at times in any type of acidosis, especially in the form due to starvation. The hydrogen ion concentration of the urine has but slight clinical significance. The reactions before and after the alkali tolerance test are misleading unless the excretory capacity of the kidneys is known to be normal. This test is therefore available only in selected cases. It may be made either by the mouth or intravenously. Five grammes of sodium bicarbonate are taken by the mouth in a moderate quantity of water every two or three hours, the urine being voided beforehand in each instance. Samples of urine not clearly acid are to be boiled in order to convert bicarbonate into carbonate which reacts readily to litmus. In the case of gastro-intestinal disease or when large doses of bicarbonate are indicated intravenous injection may become necessary. Stringent precautions are required in sterilization to prevent excessive formation of the carbonate.[1]

Acidosis plays a very important rôle in clinical pathology. In varying degrees of intensity it occurs in starvation, especially after sudden deprivation of food; in persons on a protein-fat diet, without carbohydrates, particularly when carbohydrates have been abruptly withdrawn; in diabetes mellitus, the nephritides, cardiorenal disease, cholera, pneumonia, the infectious fevers and grave septic states; in surgical anæsthesia and eclampsia. It is much more frequent in children than in adults, being common in diseases characterized by severe diarrhœa and vomiting and in the infections. The more severe forms of acidosis are described as acid intoxication, and are accompanied by the presence of acetone bodies in the urine. These substances, however, may occur in the urine in the absence of the ordinary signs of acidosis—diminution in the tension of the carbon dioxide content of the alveolar air, deepened respiration at an increased rate, increased acidity of the urine and alkali tolerance, that is, failure to produce an alkaline reaction of the urine by the administration of five grammes of sodium bicarbonate.

Although five grammes is the quantity of sodium bicarbonate sufficient to cause the urine of a normal individual to show an alkaline reaction, a much greater amount may fail to do so in persons wholly free from clinical symptoms of acidosis. With a tolerance of seventy-five grammes or more dyspnœa

[1] Consult Sellard's "The Principles of Acidosis and Clinical Methods for Its Study," 1917.

appears; with one hundred and fifty grammes, air hunger. It is therefore important that the carbon dioxide test of the alveolar air or the alkali tolerance test should be employed when severe symptoms occur under conditions which favor the development of acidosis even in the absence of the signs of that syndrome, in order that in so far as possible its special causes may be removed. It is fortunate that the alkali tolerance test is at the same time a therapeutic measure. In the severe acidosis of diabetes Joslin recommends the free administration of hot liquids, such as water, thin broths, tea to the extent of 1000 c.c. in the course of every six hours, cleansing enemata, and the withdrawal of fats from the diet. Small amounts of carbohydrates are to be given and alkali omitted or withdrawn.

Dehydration

Litchfield (1918)[1] has recently called attention to the important part played in the symptom-complex of many serious diseases by the deprivation of water. His clinical studies are based upon the laboratory researches of Erlanger and Woodyatt (1917)[2] and Wilder and Sansum (1917)[3]. After a brief review of the well-known facts concerning water from the viewpoints of anatomy and physiology, he proceeds to enumerate the symptoms resulting from the gradual or rapid deprivation of water in disease. The clinical picture is a familiar one, being made up of phenomena due to the specific effects of the primary infection, the toxæmia of retained waste products and nitrogen starvation. It is, however, obvious that the deficiency of water is of fundamental importance. The following symptoms are characteristic: Rapid respiration, a feeble, thready pulse of high frequency, a systolic blood-pressure of 70 or 60. The tongue is parched and protruded with difficulty; the surface is dry and cool and the skin shrivelled, especially upon the extremities. The features are drawn and pinched, the eyeballs sunken, the intra-ocular tension lowered. There is diminished secretion of urine, sometimes complete suppression. Constipation is common, though diarrhœa frequently occurs. The mental condition varies from more or less complete apathy, to restlessness and irritability passing into low, muttering delirium and coma.

The water equilibrium may be deranged by diminished intake or increased output. In extreme cases both may be operative. The intake may be reduced in consequence of nausea, immediate rejection by vomiting or by morbid conditions of the mouth, throat or œsophagus which prevent swallowing, or of the gastro-intestinal tract which prevent absorption. Finally apathy, delirium or coma may render it impossible for the time being to administer sufficient fluid. As the taking of food is practically impossible, a starvation acidosis of acute and dangerous type is likely to occur.

A dangerous loss of fluid may be caused by persistent vomiting, profuse diarrhœa, excessive perspiration or hemorrhage. Rapidly forming inflammatory exudates into the serous cavities constitute an important cause of dehydration, in particular when such effusions are repeatedly aspirated.

The above-described water starvation is seen in many grave infectious

[1] Glucose Therapy, The President's Address, Trans. Sect. on Pract., A.M.A., 1918.
[2] Intravenous Glucose Injections in Shock, Jr. A.M.A., Oct. 27, 1917, p. 1410.
[3] A Glucose Tolerance in Health and Disease, Arch. Int. Med., Feb., 1917, p. 311.

diseases and is frequently the forerunner of death. Among the conditions in which it is especially liable to occur are fevers of the typhoid group, sepsis, especially postoperative and puerperal sepsis, peritonitis, the meningitides and cerebral abscess, the pneumonias, empyema, pleural effusion and Shiga dysentery. The most striking forms of acute dehydration are seen in large hemorrhages and in cholera Asiatica and severe cases of cholera nostras.

The condition of dehydration closely resembles surgical shock and has been treated among other measures with some degree of success by the attempt to anticipate its occurrence or to restore the lost fluid by injections of physiological salt solution—enteroclysis, hypodermoclysis and the Murphy drip. It is a result of the experimental use of glucose in the treatment of artificially produced shock in animals that its employment in the treatment of dehydration in human beings has attracted attention. "On theoretical and experimental grounds supported by some clinical evidence, it would appear that intravenous injections of glucose at appropriate rates are of distinct benefit in certain phases of shock" (Erlanger and Woodyatt)[1]. The prognosis in a condition frequently terminal has been so often favorably influenced by the intravenous injection of suitable amounts of hypertonic glucose solution that it is referred to in this connection. Among the effects are improvement in the general appearance of the patient, the clearing of the facies, slowing of the respiration and pulse, a rise in the systolic blood-pressure, a moistening of the tongue and mouth. The kidneys and bowels resume their functions and the mind clears. The patient asks for water and food and presently may fall asleep.

The rate of administration is 0.8 gramme per kilogram of body weight per hour of a 25 per cent. solution. Rigorous sterilization of the water, glucose and apparatus is to be observed. For details of the technic consult the articles referred to in the footnotes.

Functional Tests.

The exact determination of the efficiency of the so-called vital organs, the liver, kidneys, pancreas, heart, and ductless glands would be of the highest importance in the diagnosis of the early periods of disease affecting these organs, the recognition of atypical cases and prognosis. Much of the work thus far accomplished in this field of clinical pathology is inconclusive and much is unsuited to ordinary bedside purposes. The following tests are of practical importance.[2]

TESTS OF LIVER FUNCTION

The Glycogenic Function.

The Carbohydrate or Sugar Tests.

Under certain conditions glucose is not retained in the cells but passes directly into the blood, causing hyperglycæmia. Under these circumstances it is excreted in the urine more or less continuously and in varying amounts.

[1] Loc. cit.

[2] Consult also Barton. Manual of Vital Functions, Testing Methods and Their Interpretations. Boston. 2nd Ed. Badger, 1917.

1. **The Cane Sugar Test.**—One hundred and fifty or 200 grammes of cane sugar syrup are taken fasting. The urine is collected at intervals and examined by suitable tests. See page 301 *et seq.* The glucose reaction renders the test positive. This test is invalidated by the fact that there are no methods by which the completeness of the conversion of cane sugar into glucose by the action of the intestinal juices can be known.

2. **The Glucose Test.**—One hundred and fifty grammes of dextrin-free glucose in 300 c.c. of water are administered to the fasting patient in the morning. The urine collected at intervals of an hour in separate vessels is tested for glucose. A positive reaction is not conclusive and the test should be repeated at intervals of some days, due regard being paid to the permeability of the kidneys and the possibility of spontaneous glycosuria after meals.

3. **The Levulose Test.**—(Honey may be used as a substitute).—One hundred grams of levulose is taken fasting and the urine, voided at intervals of four hours, is examined by the fermentation test or the polariscope. See page 304. This quantity should not cause levulosuria in a normal person. Much was expected from this test because levulose is not changed during digestion as it is absorbed as such. It has, however, shown in practice no advantages over the other sugar tests for functional efficiency of the liver.

4. **The Galactose Test.**—Forty grammes of galactose dissolved in 400 c.c. of water or tea taken on an empty stomach in the morning. The urine is collected at intervals and tested for sugar. See page 303. In catarrhal jaundice the reaction for sugar is fairly constant.

The sugar tests yield positive reactions in cirrhosis, icterus gravis, and cholelithiasis and in greater frequency in catarrhal than in obstructive jaundice; but they are complicated by unknown factors among which intestinal absorption and renal permeability are the most obvious.

The Ureagenetic Function.

Most of the tests for derangements of this function of the liver are so complicated that they cannot be carried out except by an expert chemist, and there are many undeterminable physiological factors which serve to modify the results. For these reasons this series of tests are not available for ordinary clinical work.

The Antitoxic Function.

The tests for the integrity of this function of the liver depend upon the fact that poisons reaching the organism by way of the portal circulation are arrested and destroyed by the liver cells.

1. **The Methylene Blue Test.**—Inject 5 c.c. of a 5 per cent. solution of methylene blue subcutaneously. Collect and examine the urine at first in half an hour, subsequently at hourly intervals. Normally it is colored at the first examination, the coloration attaining its maximum intensity in three or four hours and disappearing in about forty-eight hours. If the elimination is not continuous but intermittent, the test is positive as indicating insufficiency of the hepatic cells.

2. **Roche's Modification.**—The methylene blue .002 gm. is given by the mouth in capsule in the morning on an empty stomach. The urine is collected every four hours in separate vessels. If the second voiding is colored, the inability of the liver cells to arrest the pigment is established. The urine of the later voidings is colored green. The permeability of the kidneys must be previously ascertained. See page 309.

3. **Indicanuria as a Test of the Fixation Function of the Cells.**—It has been assumed that the normal liver is physiologically capable of destroying the indican which is formed in the intestine as the result of the putrefaction of albuminoids. It would follow that the spontaneous presence of indican in the urine is evidence of impairment of the function of the hepatic cells.

Provocative Indicanuria.—The patient is restricted to an exclusive milk diet for a few days. One-thousandth gm. of indol is administered on an empty stomach. The urine is collected every four hours and examined for indican. A positive result indicates impaired hepatic function.

Obermayer's Test.—See page 293.

The Sanguinopoietic Function.

Estimation of Blood Coagulation Time.

1. **Wright's Method.**—See page 256.

2. **The Fibrinogen Test.**—A rough estimate of the fibrinogen content of the blood may be made by coagulating a little plasma with calcium and testing the firmness of the clot with a glass rod.

3. **Whipple Horwitz Method.**—Fibrinogen is precipitated by subjecting 20 c.c. of oxalated plasma to a temperature of 59° C. for twenty minutes. The precipitate is separated by centrifugation, washed with sterilized water, alcohol and ether, dried at 120° C. and weighed. The normal fibrinogen content of the blood plasma is .30 to .40 gm. to 100 c.c. The proportion is diminished in general lesions involving the parenchyma of the liver. In cirrhosis it is frequently low.

4. **The Fibrinolysis Time.**—The clot of normal blood remains undigested for some days or weeks. It has been suggested that the dissolution of the clot is caused by the action of an enzyme. In cases of cirrhosis the blood digests the clot in a few hours at body temperature. The activity of the enzyme in question is destroyed by heat and inhibited by normal serum.

The Goodpasture Test.—Blood is obtained from a vein in the arm and the coagulation time is estimated. A portion of the blood is drawn into 1 per cent. solution of sodium oxalate to prevent clotting. This specimen is centrifugated and the fibrinogen content estimated. Portions of the clot and specimens of oxalated serum are placed in the thermostat at 37° C. They are examined at intervals of an hour. If the test is positive the clot liquefies and is wholly dissolved in three and one-half to five hours; if negative there is no dissolution of the clot for several days.

The Biliary or Exocrinous Function.

1. **Tests for Biliary Pigments.**—See page 294.

TESTS OF KIDNEY FUNCTION.

The Water Tests.

1. **Provocative Polyuria Test.**—The morning urine is measured and its specific gravity taken. Its total sodium chloride and urea are to be estimated. The patient then drinks 500 c.c. of ordinary or mineral water. The urine is collected every half hour by voiding or catheter, if the general renal function is to be studied; or by ureteral catheterization if the function of each kidney is to be compared. The maximum polyuria is reached under normal conditions at the end of the first half hour, and the total solids diminish. If the renal function is impaired the polyuria is delayed or does not occur.

2. **The Strauss-Grünwald Test.**—The patient takes no drink or food after 7 P.M. At 6.30 A.M. the night urine is collected and he drinks 500 c.c. of water. The urine voided at 7, 8, 9, 10 and 11 A.M. is collected and the respective quantity and specific gravity of each portion determined. During this whole period the patient rests quiet in bed or upon a couch. Normally the amount voided equals that taken, at the beginning, so that by 10 o'clock he will have passed a pint. About 8 A.M. the specific gravity is at its lowest. Variations in the amount, time and specific gravity point to derangement of the renal function.

Sodium Chloride Tests.

1. **Mohr's Method.**—See page 292.

2. **A rough estimate** of the sodium chloride content of the urine may be made by adding to a test tube of clear urine free from albumin ten drops of pure HNO_3 and one drop of 1 to 8 solution of $Aq.NO_3$. If chlorides are normal or increased a precipitate forms as a compact ball which slowly sinks: if diminished, the globular mass of precipitate is less compact; if greatly diminished, it forms a cloud without solid flakes.

Estimation of Urinary Nitrogen.

The kidney does not play any part in nitrogen metabolism. Its function is to excrete nitrogenous waste products brought to it in the blood current. Urinary nitrogen as an index of renal function is represented by the amount of urea eliminated under normal conditions, the patient being on a fixed diet and pursuing the same course of life, with an estimate of the power of the kidneys to eliminate more urea, when the proteid intake is increased or urea itself is ingested. The ordinary routine estimation of the amount of urea in the urine without the precaution of a fixed regimen is insignificant. If under proper preeautionary measures the constant approximately normal output of urea, about 30 to 34 grammes in twenty-four hours, is maintained, the renal function of the kidneys may be assumed to be equal to the physiological requirement of the individual. This is especially true if, the proteid intake being increased, the urea output shows a prompt corresponding rise.

1. **Marshall's Method of Urea Estimation.**—This method depends upon the conversion of urea into ammonium carbonate by the action of an

enzyme prepared from soy bean. This enzyme, in a form suitable for immediate use and called urease, is prepared in the Hynson, Westcott and Dunning Laboratory. To estimate the amount of urea in urine, determine with decinormal hydrochloric acid and methyl orange the degree of natural alkalinity of a portion of the specimen treated with the enzyme and compare it as to alkalinity with an equal quantity of the same specimen not so treated. The difference represents the ammonium carbonate formed by the breaking up of the urea present. The amount of urea is ascertained by calculating its equivalent in ammonium carbonate. See also page 290.

2. **Ambard's Urea-Coefficient.**—This investigator, as the result of his comparative simultaneous studies of the urea-content of the blood and the urine in normal subjects, concluded that (1) when the concentration of urea in the urine is constant, the excretion varies directly as the square of the concentration of urea in the blood, and (2) when the concentration of urea in the blood remains constant, the excretion varies inversely as the square root of the concentration in the urine, and (3) that other factors being the same, the excretion varies directly with the weight of the individual. These conclusions are known as Ambard's laws, the mathematical formula by which they are expressed as Ambard's coefficient, and the constant is indicated by the symbol "K," the value of which is in normal human beings 0.08, but in cases of renal disease attended with an increase in the concentration of blood urea and a decrease of the elimination of urea in the urine the value of "K" is greatly increased. This method of renal function testing is not available for the practitioner.[1]

3. **Provocative Urea Test.**—Thirty grammes of urea dissolved in a small tumblerful of water are given with a light breakfast consisting of gruel or some cereal. Additional water is taken afterwards to induce diuresis. The urine is collected two hours before the breakfast as a standard for comparison and every two hours afterwards for twenty-four hours, and the urea content of the different specimens determined at the end of the period. Normally there is a marked rise in urea excretion on the second two-hour period. The absence or delay of such a rise indicates deficiency of kidney function. This test is invalidated by gastric stasis or delay absorption from any cause.

Dietary Test of Kidney Function. Twenty-four Hour Test Meals for Nephritic Function.[2]

This functional test, originally suggested by Hedinger and Schlayer (1914) and fully worked out by Mosenthal (1915), has proved of great importance in the clinical study of renal function and come rapidly into general use. It has yielded practical results in the diagnosis of the forms of kidney disease, various cardiopathies and other conditions attended with derangements of urinary output and dropsical states. The

[1] "If we decline to consider the kidney a fixed and unchanging valve in the bottom of a cylinder of blood, and remember that it is of the same fundamental substances and subject to the same laws that apply to blood, then we cannot be so exacting in our demand for mathematical relationship between the blood urea and urine urea. Since this organ of excretion is itself so largely composed of blood, it seems obvious that its function cannot be expressed by a constant but will depend on the physico-chemical changes that may occur in the blood. There may, therefore, be shifts in the constituents of the blood that will not be mirrored by the urine." Atchley, Arch. Int. Med., vol. 22, No. 3, 1918.

[2] Arch. Int. Med., 1915, xvi, p. 733 *et seq.*, and Barton, p. 97 *et seq.*

urinary water and specific gravity are accurately determined at intervals of two hours. The salt and nitrogen are estimated for the day and night specimens.

The following schedule contains the directions for the test meals:

DIET

TEST MEALS FOR NEPHRITIC FUNCTION

For ..... Date.....

All food is to be *salt-free* food from the kitchen.

Salt for each meal is to be furnished in weighed amounts. One capsule of salt, containing 2.3 gm. sodium chloride, is furnished with each meal. The salt which is not consumed is returned to the laboratory, where it is weighed, and the actual amount of salt taken calculated.

All food or fluid not taken must be weighed or measured after meals, and charted in the spaces below.

Allow no food or fluid of any kind except at meal times.

Note any mishaps or irregularities that occur in giving the diet or collecting the specimens.

BREAKFAST, 8 A. M.

Boiled oatmeal, 100 gm.	Coffee, 160 c.c.
Sugar, ½ teaspoonful.	Sugar, 1 teaspoonful.
Milk, 30 c.c.	Milk, 40 c.c.
Two slices bread (30 gm. each).	Milk, 200 c.c.
Butter, 20 gm.	Water, 200 c.c.

DINNER, 12 NOON

Meat soup, 180 c.c.	Butter, 20 gm.
Beefsteak, 100 gm.	Tea, 180 c.c.
Potato (baked, mashed or boiled), 130 gm.	Sugar, 1 teaspoonful.
	Milk, 20 c.c.
Green vegetables as desired.	Water, 250 c.c.
Two slices bread (30 gm. each).	Pudding (tapioca or rice), 110 gm.

SUPPER, 5 P. M.

Two eggs, cooked any style.	Sugar, 1 teaspoonful.
Two slices bread (30 gm. each).	Milk, 20 c.c.
Butter, 20 gm.	Fruit (stewed or fresh), 1 portion.
Tea, 180 c.c.	Water, 300 c.c.

8 A. M.—No food or fluid is to be given during the night or until 8 o'clock the next morning (after voiding), when the regular diet is resumed.

Patient is to empty the bladder at 8 A. M. and at the end of each period, as indicated below. The specimens are to be collected for the following periods in properly labelled bottles:

8 A. M.–10 A.M., 10 A. M.–12 N., 12 N.–2 P. M., 2 P. M.–4 P. M., 4 P. M.–6 P. M., 6 P. M.–8 P. M., 8 P. M.–8 A. M.

The diet subjects the kidneys to a certain amount of temporary stress since it contains substances which act as diuretics. It contains about 13.4 gm. nitrogen, 8.5 gm. salt, and 1760 c.c. water, with purin material in the soup, meat, tea and coffee. It is essential to the test that the urine be collected exactly at the stated intervals, that no food or fluid of any kind be taken between meals or during the night and that the complete twelve-

hour night specimen be fully voided before breakfast. These precautions are necessary in view of the promptness with which the kidneys respond to the ingestion of fluid.

The urine of healthy subjects undergoing this dietary test shows:

1. Variations in the specific gravity of the two-hour specimens reaching nine points or more. The variations are less when the amount of fluid is reduced or there is diminished urinary secretion.

2. An approximate balance between the output and intake of salt, nitrogen and fluids.

3. A night urine of high specific gravity, 1016 or more; moderate amount not exceeding 400 c.c. and uninfluenced by the amount of fluid ingested or urine voided during the day.

It is a function of the kidney in health to concentrate or dilute the urine readily and thus maintain the normal concentration of the fluids of the body. Failure of this function shows itself in fixation of the specific gravity. This sign is therefore an indication of disease which may primarily reveal itself as nephritis or as extra-renal and secondarily impairing the function of the kidney, as cystitis, pyelitis, hydronephrosis, renal congestion of cardiac origin, diabetes or anæmia.

An increase of the amount of night urine beyond 400 c.c. is an early symptom of nephritis and may precede by some time distinct albuminuria and the presence of casts.

The Response to the Dietary Test in Disease.—*Chronic Interstitial Nephritis.*

1. Fixed and low specific gravity.
2. Diminished output of salt and nitrogen.
3. Polyuria.
4. Night urine differing from the normal is increased in amount; low specific gravity and low nitrogen concentration.

These responses vary with the grade of the pathological changes in the kidney and become very marked in advanced cases. Similar responses to the dietary test are encountered in disease of the urinary passages, as prostate enlargement, cystitis, ureteritis and pyelitis; anæmias of high grade and in certain diseases of the kidney other than the nephritis, as pyelonephritis and polycystic kidneys.

Renal Congestion from Myocardial Insufficiency.

1. Specific gravity fixed, usually about 1020.
2. Low output for salt.
3. A sufficient nitrogen output.
4. Persistent diminished urinary water.
5. Normal night urine.

During the elimination of the œdema:

1. Low specific gravity tending to fixation.
2. Nitrogen normal.
3. Salt output in excess of intake.
4. Water elimination greater than intake.

After the œdema has disappeared the response to the dietary test still indicates impaired renal function as shown by condition resembling those in interstitial nephritis.

1. Specific gravity low and tending to fixation.
2. Normal nitrogen and water.
3. Salt output somewhat diminished.
4. Night urine variable in amount. It may or may not be increased.
5. Nitrogen concentration low.

Chronic Parenchymatous Nephritis.—During the period of œdema the test response is:

1. High specific gravity.
2. Salt and water retention.
3. Nocturnal polyuria.
4. Good nitrogen elimination.

With the disappearance of œdema the response is similar to that in cases of myocardial insufficiency under the same conditions.

The Blood in the Estimation of Renal Function.

Impaired renal function results in the accumulation of the nitrogenous products of metabolism in the blood. It follows that marked accumulation of incoagulable nitrogen or of urea is evidence of renal insufficiency. In cases of nephritis such accumulation is an ominous prognostic sign.

1. **The determination of the urea** in the blood may be most rapidly and accurately made by the method of Marshall, while the technic of estimating total incoagulable or residual nitrogen in the blood serum yields satisfactory results by Morris' modification of the Hohlweg-Meyer plan or that of Folin and Denis. These tests require a well-appointed laboratory and are impracticable for the general practitioner.

2. **The blood coagulation time** as a test for renal function requires 20 c.c. of blood and is not available for general use.

3. **Cryoscopy** has not come into general use. See page 308.

The Elimination of Foreign Substances by the Kidneys as a Test of Function.

1. **Potassium iodide, phloridzin** and **hippuric acid** have been used for this purpose, but their employment in the clinic has been abandoned.

2. **The Lactose Test.**—It has been experimentally established that this substance may be regarded as an index of the vascular and glomerular function of the kidney. The technic is as follows: Two and five-tenths grams of pure lactose are dissolved in 25 c.c. of freshly distilled water and pasteurized for four hours upon four successive days at a temperature of 80° C. A fresh solution must be prepared for each injection and the details of the technic for intravenous injection carefully observed. Occasionally there is constitutional reaction, but this occurrence is unusual. The urine voided at the end of four hours and at hourly intervals thereafter is tested for sugar by Nylander's reagent. (See page 302.) The test depends upon the time. Over six hours indicates renal insufficiency.

Elimination of Dyes by the Kidneys.

1. **Methylene blue** and **indigo carmine** have been extensively employed as tests of renal permeability, the time of first appearance, that of maximum intensity and that of total excretion were noted and formed the basis of deductions concerning the function of the kidneys. Their use for this purpose has been almost completely superseded by the phenolsulphonaphthalein test of Rowntree and Geraghty.

2. **Phenolsulphonaphthalein Test.**[1]—Twenty minutes to half an hour before giving the test the patient is given 200 to 400 c.c. of water to insure diuresis. The bladder is completely emptied. The time being noted, 1 c.c. of a solution of the drug is injected into the lumbar muscles. The solution is prepared as follows: .6 gm. phenolsulphonaphthalein and .84 c.c. double normal NaOH are added to .75 per cent. NaCl solution up to 100 c.c. Add two or three drops of double normal NaOH. The color becomes Bordeaux red and the solution is non-irritant.

The urine is passed into a test tube containing a drop of 25 per cent. NaOH and the time of appearance of the first pinkish color noted.

If there is no urinary obstruction the catheter is not necessary after the appearance of the color, and the patient may then retain the urine and urinate at the end of one hour in one receptacle and again at the end of the second hour in another.

A rough estimate of the time of the appearance of the drug in the urine may be gained by having the patient urinate frequently a small amount without the catheter. In prostate cases it seems better to keep a catheter *in situ*. If this is done the catheter may be corked and this is removed at the end of the first and second hours.

Each sample of urine is measured. Twenty-five per cent. solution NaOH is added to make the color maximum. The urine is usually yellow or orange and becomes deep purple on addition of the alkali. The solution is put in a liter flask and diluted with distilled water to make a quart. This is thoroughly mixed and a portion is filtered and compared with a standard in a colorimeter. The standard solution consists of .003 gm. phenolsulphonaphthalein (½ c.c. of solution used for injection) diluted to 1 liter and made alkaline with a few drops of 25 per cent. NaOH. The test solution retains its fine purplish color for a week or more.

The colorimeter contains a wedge-shaped cup which is filled with the standard solution. The rectangular cup is filled with the solution to be tested. The wedge-shaped cup is manipulated by a screw until the color fields are identical. The percentage is read off on the indicator scale.

Technic of the Phenolsulphonaphthalein Test as Applied to Estimation of the Function of the Individual Kidney.—Twenty minutes previous to the application of the test the patient is given 600 to 800 c.c. of water to provide a free flow of urine. The ureters are catheterized, a special catheter being recommended, namely, the flute end catheter of Albarran No. 6 or No. 7. The catheters are passed four inches into the ureters. The cystoscope is withdrawn, leaving the catheters *in situ*. A small urethral catheter is passed into the bladder to empty that organ and

[1] Barton: Manual of Vital Function Testing Methods and Their Interpretation. 2nd edition. Rowntree and Geraghty: Arch. Int. Med., 1912, ix, 284.

detect later leakage. The other details of the test are similar to those of the ordinary technic (*q. v.*).

"Where only one test can be employed the most value is unquestionably to be obtained from the usé of phthalein: and this is particularly so from the standpoint of the surgeons." Geraghty.

TESTS OF PANCREATIC FUNCTION.

The enormous importance of the pancreas as an organ of digestion and as a gland of internal secretion stands in strong contrast to our ability to determine its functional efficiency in either of these respects by clinical or laboratory methods. Except in the case of gross changes such as are associated with acute pancreatitis, certain forms of pancreatic diabetes and manifest lesions as tumor or cyst, it is exceedingly difficult for the clinician to recognize the presence of disease of this organ or determine the character and extent of the pathological process. In view of the manifold and complex nature of its physiological functions and the difficulties which beset their experimental study, the unsatisfactory results of efficieney tests are not surprising.

Tests of Functions of External Secretion.—1. **The Examination of the Fæces.**—The stools in which evidences of impairment of the pancreatic functions of digestion are present—as an excess of muscle fibres, undigested nuclei, excess of fat and split fats and an excess of undigested starch granules—are usually very bulky. If formed the cylinders are of abnormal diameter. The total dried fæces may weigh 150 grammes instead of the normal average of about 50 grammes.

The ordinary methods of examination should be preceded for three days by Schmidt's diet, which contains inconsiderable amounts of all three varieties of food elements and is therefore well suited to all tests which require an examination of the faces. See pages 226 to 232.

2. **Test for Pancreatic Ferments in the Stools.**—Methods for the qualitative and quantitative estimation of trypsin, diastase and the liptolytic ferment in the fæces are to be consulted in the manuals of laboratory technic but they are of little practical value for clinical purposes.

Tests of the Function of Internal Secretion of the Pancreas.—These are three:

1. **The Cammidge Reaction.** (See page 308.)

2. **Spontaneous and Provocative Alimentary Glycosuria.**—Glycosuria cannot be regarded as an indication of disease of the pancreas, since it may have its origin in disease of any of the so-called diabetogenous organs. This group includes the kidney, the liver, the thyroid, the chromaffin system, the hypophysis and the central nervous system. The concurrence of glycosuria and the evidences of insufficiency of the external pancreatic secretion warrants the hypothesis that the diabetes is due to pancreatic disease. That such is, however, the case, cannot be positively affirmed for the reason that cases of pancreatic diabetes may occur in disorder of the internal secretion without evidence of insufficiency or absence of the external secretion.

3. **The Pupillary Test of Pancreatic Insufficiency.**—Loewi discovered

that after the removal of the pancreas in laboratory animals the instillation of adrenalin causes dilatation of the pupil. This phenomenon he attributed to an increase of the action of the sympathetic system resulting from the absence of the inhibitory influence of the internal secretion. Under normal conditions adrenalin does not produce mydriasis when injected into the conjunctival sac, although it does so when injected intravenously. The test is made by the instillation of 3 or 4 drops of 1:1000 solution of adrenalin into the eye. Mydriasis comes on slowly, reaching its maximum in 30 to 60 minutes.

TESTS OF THE FUNCTIONAL CAPACITY OF THE HEART.

The physiology of the heart is extremely complex. Englemann enumerated the following fundamental attributes of the cardiac musculature: (1) The power of originating contractile impulses—the dromotropic function; (2) the capacity of response to stimuli; excitability—bathmotropic function; (3) the faculty of conducting impulses, conductivity—the dromotropic function; (4) the property of contractility—the inotropic function; and finally (5) the vital function of tonicity in consequence of which the chambers of the normal heart maintain their size during diastole.

The normal interrelation and harmonious exercise of these functions result in the maintenance of the circulation by the regular alternation of the cardiac systole and diastole at the rate in healthy adults of from 60 to 80 revolutions to the minute; in infants 90 to 140; in the aged 70 to 90. Extreme variations in rate and power occur in response to the requirements of the individual in health and as the result of diseases. Those in health are as a rule transient; while the departures from the normal rhythm and force which result from disease are more or less continuous, and those due to pathological changes in the heart or blood-vessels are usually persistent. It is of the mechanism of the heart to adjust itself within wide limits to the constantly changing needs of the individual. The normal property of response to the demand for more power has been spoken of as "the reserve power" of the heart, and the capacity of continuous adjustment to the advancing requirements of valvular lesions which constitutes compensation is attended by progressive impairment of this reserve. The recognition of the more advanced degrees of failure of compensation is among the simplest problems of clinical medicine: there are no tests by which the beginnings of failure may be determined. The recognition of the early stages of functional impairment of the heart constitutes a legitimate subject of laboratory research, but the results both as regards alike the fundamental functions and as regards the absolute power of the heart are thus far unsatisfactory and the practitioner must content himself with the older clinical methods and such results as attend the present-day use of such instruments of precision as the polygraph, sphygmodynamometer, electrocardiograph and the röntgen-rays.

There are four general groups of tests of cardiac function:

1. Those based upon the reaction to active or passive muscular movements.

These tests depend upon the influence upon cardiac function, as mani-

fest by changes in the pulse, blood-pressure, and in the area of precordial dulness, of various exercises, changes of posture, compression of peripheral arteries by the use of a tourniquet or bandage, resisted movements, holding the breath, elevation of the arms, and so forth.

2. Those based upon cardiac reflexes.

This group includes (a) energetic friction over the heart. If the reflex is normal the area of cardiac dulness is diminished.

(b) The test consists in making a series of strong strokes over the median line of the abdomen. A positive result consists in an increase of cardiac dulness to the right.

3. Sodium chloride elimination as a test of cardiac function.

A constant daily quantity of sodium chloride is given for some days. An equilibrium being determined, the daily quantity of sodium chloride is doubled and a quantitative estimate of the salt in the urine is made. In functional cardiac inadequacy, even though latent, the salt elimination is defective and there may be œdema. The permeability of the kidneys must be determined beforehand.

4. Instrumental methods.

(a) The sphygmomanometer (see page 124).

(b) Sphygmobolometry and sphygmobolography.[1]

(c) Energometry.[2]

(d) Electrocardiography (see page 112).

In conclusion, the following quotation from Hirschfelder[3] is pregnant with wisdom for the practitioner:

"It must be admitted that in order to be decisive, all tests have to be pushed to a point at which the appearance, sensation and signs of the patient are in themselves perfectly characteristic of cardiac insufficiency, and at which, for diagnostic purposes, a little common-sense observation is at least as unambiguous as observation with elaborate apparatus. This does not mean that exercise tests are unimportant. On the contrary, they are of the greatest value and no change in the patient's mode of living during convalescence or during after-life should be undertaken without them.

"But their importance depends more upon the care with which the physician watches the general appearance and condition of the patient, the rapidity with which he recovers from the exercise, his general condition and whether nervousness, irritability, cough or insomnia have set in during the twenty-four hours following it, than in the numerical changes which occur at the moment of exercise. The symptoms to be looked for as evidence of overwork are well known. These are subtler manifestations resulting from smaller changes than may be detected by even the most refined observation by mechanical methods and which are less easily masked by ambiguities.

"Moreover, it must be realized that any one form of exercise furnishes data which may depend as much upon the condition of the skeletal muscles as upon the heart. The blacksmith with a diseased heart may be able to do

[1] See Barton l. c. p. 243 *et seq.*, p. 251 *et seq.*
[2] See Barton l. c. p. 253 *et seq.*
[3] Diseases of the Heart and Aorta, 3d ed., J. B. Lippincott Company, Phila., 1918, p. 211, 212.

more work than the bookkeeper with neurasthenia, and yet under the conditions in which he lives, even if not under the strength test arranged for the average man, the blacksmith's heart may be failing. The important question is not what the person can do in a gymnasium, but what he can do and what he cannot do in everyday life. Each man must be fit for his own mode of life or must be made to change it. His cardiac power must be studied with reference to that mode of life rather than with reference to a rigid scheme."

ENDOCRINE GLANDS.

The opinion prevalent among physiologists until recent years that the chemical processes of the body were confined to the building up and breaking down of various tissues, absorption, combustion, exertion and analogous processes has undergone remarkable changes. It is now recognized that the functions of the body are regulated not only by nervous influences but also by chemical substances that are elaborated by certain glandular excretions and discharged into the blood stream. These substances originate in gland-like organs that have no discharging ducts, as the thyroid, the parathyroid, the thymus, the adrenals, the hypophysis, and the epiphysis—the glands of internal secretion. They also are elaborated by certain other glands, which possess the additional function of secreting a fluid which is discharged through the ducts—external secretion. The glands which possess this double function include among others the liver, the pancreas, the intestinal mucosa, and sex glands. These internal secretions exercise a profound influence upon the growth, development and metabolism of the body, and play a prominent part in many pathological conditions which arise in consequence of the hyperfunction, hypofunction, or dysfunction of the respective glands, derangements of their reciprocal relations and aberrant combinations, such as arise in the so-called polyglandular syndromes. (See Diseases of the Ductless Glands, Vol. II.) Practical tests of the functions of these most important organs would be of the highest value to the clinician, but despite the elaborate investigations thus far made, are not yet available, a fact due partly to lack of exact physiological knowledge of the subject and partly to the difficulties which attend the finer research in metabolism.

Perhaps the sole outcome available for everyday medicine is the therapeutic test for impaired functional efficiency of the thyroid gland. If in a suspected case, thyroid extract be administered in proper doses, the prompt disappearance of the symptoms and remarkable improvement in the general condition of the patient constitute a positive reaction.

Serology.[1]

In the study of the immune reactions of the blood-serum and the accompanying changes found in the cerebro-spinal fluid, we have a series of examinations which are an invaluable aid to the diagnosis of diseases of the central nervous system. These tests provide a way of differentiating between functional and organic conditions both in neurology and psychiatry, and in

[1] Contributed by Corson White, M.D., as collaborator.

syphilis they yield not only a means of early diagnosis but act as a guide for treatment. Experience has shown that a diagnosis should in no case be decided upon the result of one test, but all examinations of proven values should be investigated and the correct interpretation be made upon the combined results of the laboratory findings and careful clinical studies.

Many important facts concerning the cerebro-spinal fluid were demonstrated before Quincke (1891) published his observations on the technique and value of lumbar puncture. No decisive studies of the fluid in mental or nervous diseases were reported, however, until 1901, when the investigations of Ravaut and Sicard, and later, Widal appeared. Since then, innumerable researches—bacteriological, chemical, serological and morphological—have been published, covering syphilis in all its many manifestations and all types of nervous and mental diseases. These observations have placed in the hands of clinicians a mass of material, most of which is of immediate value.

The removal of the spinal fluid is a relatively simple procedure and often of profound significance for both patient and physician. No elaborate preparation is necessary. Two (2) glasses of water given just before the puncture and free evacuation of the bowels will usually prevent the headache which often follows the withdrawal of the fluid.

The patient may sit on a stool or edge of bed or may lie on the left side. If the recumbent position is assumed it is essential that the patient lie on a firm surface, so that the midline of the vertebral column and head is straight. Many failures, so-called dry taps, painful punctures, and withdrawal of bloody, useless fluids are undoubtedly due to attempts to puncture the subarachnoid space when the back is curved by reason of a sagging mattress. In either position of the patient the back must be arched by the bending forward of neck, head and shoulders, and the drawing up of the knees on the abdomen, the ideal position being that one which gives the greatest width between the spinous processes of the lumbar vertebræ. When the patient is in position the operator, by running his fingers along the vertebral spine, selects a soft spot between the third and fourth or fourth and fifth lumbar vertebræ. The site can be located by drawing a line between the highest part of the iliac crests across the spinal column. This line passes through the interspace between the fourth and fifth lumbar vertebræ, which is the point of election because here the space is usually the widest and there is less danger of injury to the spinal cord or sensory nerve roots.

When the site is selected, the back must be thoroughly cleansed—absolute sterility is essential. The back is scrubbed with green soap and hot water, followed by hot water, alcohol, and ether. The site is then painted with ten per cent. (10%) iodine. Many operators omit the preliminary cleansing and give, instead, three coats of iodine.

Before the actual puncture the skin may be rendered less sensitive by injection of cocaine, eucaine or by ethyl chloride spray. In some cases, notably paresis and tabes, the absence of all pain is marked. The introduction of the needle is hardly noticed and anæsthesia of any kind is not essential. General anæsthesia is rarely, if ever, needed.

The needle most commonly used is made of flexible material, preferably iridio-platinum, 10 cm. long with 1 mm. bore, supplied with a stylet which can be locked in position and is exactly flush with the sharp but short-pointed

needle. With the site selected and cocainized, the needle is inserted with a sudden thrust directly in the median line and in the centre of the interspace; straight forward into the spinous ligament, then more slowly into the canal. When the canal is reached, which is evident by the sudden give or loss of resistance, the stylet is removed. Some operators prefer the insertion of the needle (1 cm.) to side of spinal column in the interspace between fourth and fifth lumbar vertebræ, passing the needle upward and inward toward the centre. This method is of value only in fat subjects; in all others the central position is much easier and the danger of touching the bone much less.

When the technique has been properly carried out the fluid should be free from blood. It is well, however, always to collect the spinal fluid in two (2) sterile test tubes, in case the first be contaminated with blood or tissue cells. After eight (8) or ten (10) c.c. have been collected the needle should be quickly withdrawn and the place of puncture sealed with collodion.

Ordinarily, there is no danger in lumbar puncture if infection be carefully excluded and the fluid is slowly withdrawn. Occasionally, the needle may touch a nerve filament, giving rise to pain in the distribution of that nerve, or the operator may puncture the bone, causing a dull pain which may last several hours. The great majority of patients, especially paretics and tabetics, are little, if ever, affected by the removal of the fluid. There is, however, another small group of individuals, representing about seven to ten per cent. of those punctured, who suffer for a day or more from severe headache, pain in the back, and now and again have nausea, vomiting, and diarrhœa. These patients are usually those with normal fluid findings. These symptoms are not serious and are easily relieved by rest in bed until no return of the headache is noticed by the patient when sitting up. It is well to have all patients remain in bed after lumbar puncture for at least twenty-four hours.

In no sense do the symptoms recorded above constitute a contraindication for puncture. There exist, however, definite contraindications. Nonne, in an extremely large experience, reported four fatal results immediately following puncture—all cases of brain tumor. Troeme collected thirty-five fatalities from the literature, one-half of which occurred in cases of tumor of the posterior fossa. He is of the opinion that successful puncture could be performed in these cases if made with the patient in the recumbent position and the fluid be drawn out a drop at a time and limited to 2 c.c. Kaplan suggests the introduction of an equal amount of sterile normal salt solution immediately after the puncture and keeping the patient in bed with the foot raised about twelve inches.

The fluid collected from the spinal puncture should be fully examined. Experience has taught the necessity of depending in no case upon the result of one examination. No parallelism regularly exists between any abnormal finding in the fluid and any other. One cannot say that because one test is negative another will be. Conclusions must, therefore, be drawn only from a careful analysis of all the examinations of known value compared with the results of these examinations in normal fluid.

Normal cerebro-spinal fluid is a transparent water-clear liquid with a sp. gr. of 1.002 and 1.007. There may be no cells found in the examination,

and there are never more than 5 per c.cm. Occasionally, there is an isolated endothelial cell. It contains a faint trace of protein, the major part of which is globulin. It is always sterile. The Wassermann Reaction and other immune reactions are absent. The value of other immune reactions in spinal fluid is not established. A reducing substance, probably a sugar, is always present. From these normal findings a routine examination has been developed which gives most decisive information. It should include observations on pressure, transparency and color—the significance of which is described later; examination for increase in cells and for increase in globulin content—tests which always indicate the existence of organic change in the brain or cord; the Wassermann Reaction—a specific test for the detection of syphilis; and the colloidal gold reaction which differentiates between paresis and other types of syphilitic disease. The technique of these reactions can be found in any work on laboratory methods.

Abnormal Conditions.—The great majority of nervous and mental diseases yield a fluid of normal appearance. Sometimes on withdrawal, especially if collection of the fluid is made entirely into one tube, there may be a faint cloudiness due to blood-cells washed through the needle, and recognized only when they have been deposited. This is an accident of technique and can be avoided by collecting in two tubes. In case of hemorrhage into the subarachnoid space, after injury to the skull or vertebral column or following ventricular hemorrhage, a definitely bloody fluid may occur. In other conditions, as meningitis or abscess, cloudy or even purulent fluids may be found. This turbidity is due to immense increase in cells which separate out from the fluid on standing, forming a definite sediment. This sedimentation is sometimes found in fluids which on first sight seemed clear.

Turbidity occurs most frequently in bacterial infections, and should always call for a bacteriological examination. A more important discoloration is xanthochromia. This fluid has a lemon yellow color about the shade of pale blood serum. It shows a very marked increase in globulin; very often clots spontaneously and contains usually only a normal number of cells or a low pathologic count. Nonne described this condition as a spinal fluid compression syndrome. All the cases reported occurred in paraplegias or in those whose symptoms were suggestive of cord compression. This type of fluid has been observed in a pachymeningitis of the conus terminalis of syphilitic origin, in tubercular-meningitis, in Pott's disease, Landry's paralysis, pachymeningomyelitis, spinal cord tumors, extradural and intramedullary, carcinomatous metastases of the vertebra. The marked increase in globulin is probably due to stasis distal to the point of compression and not to inflammation. The color is probably due to a mixture of blood—coloring matter—from an old hemorrhage or outwandered red cells. The syndrome does not differentiate between extradural or intramedullary growths, nor give a cause for the compression. It is simply significant of compression of the spinal cord.

Pressure. —As a general rule, cerebro-spinal fluid is collected without any reference to the pressure—other than noting the rate of flow from the canula, as the puncture needle reaches the dural sac.

It is frequently advisable, however, to know exactly the height of this pressure. One may resort to the following method (Sahli) which is a modi-

fication of the method originally used by Quincke. No accurate estimation is possible unless the patient be in a horizontal position with the median line of the head on a level with the spinal canal. As soon as the needle penetrates the dura, a connection is made with a mercury manometer by means of a rubber tube filled with a one per cent. solution of carbolic acid. The portion of the manometer above the level of the mercury, forming the connection between it and the carbolic acid tube, must also be filled with the fluid. The manometer is filled with mercury to the zero point and held in such a manner that this point is on a level with the point of the aspirating needle, which is possible with ordinary manometers only when the connecting tube is of considerable length. Under normal conditions the dural pressure, in the horizontal position, ranges between 5 and 7.5 mm. of mercury or 60 to 100 mm. of water if a water manometer is used.

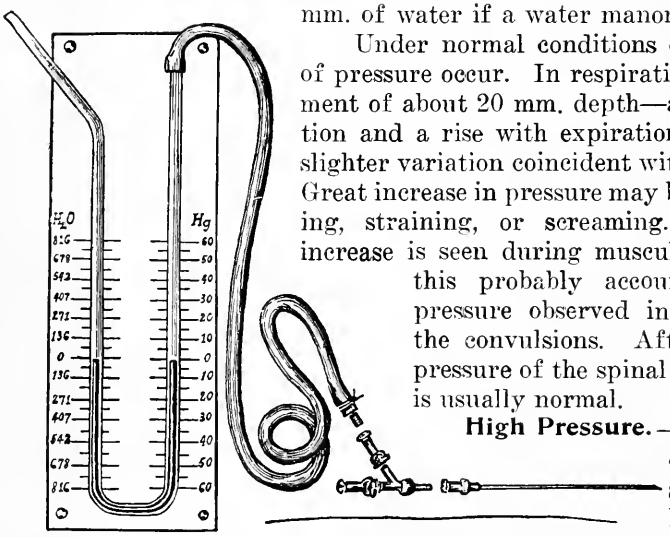


FIG. 205 *B*.—Manometer for determination of the pressure of the cerebrospinal fluid. Redrawn after Neisser. The scale shows the absolute value in mm.; the pressure measured is twice the reading. Handb. d. Neurologie I, p. 1180.

Under normal conditions certain oscillations of pressure occur. In respiration there is a movement of about 20 mm. depth—a fall with inspiration and a rise with expiration. There is also a slighter variation coincident with heart pulsations. Great increase in pressure may be caused by coughing, straining, or screaming. A still greater increase is seen during muscular resistance, and this probably accounts for the high pressure observed in epileptics during the convulsions. After the spasm, the pressure of the spinal fluid in these cases is usually normal.

High Pressure.—In pathologic conditions the pressure is frequently high. Generally, in acute disease a moderate degree of pressure increase, as registered on the manometer, is associated with severe clinical pressure symptoms, while in chronic conditions the registered increase may be much more marked with symptoms much less severe. The pressure is usually high in brain tumor, abscess, and inflammatory conditions, especially those with much exudate.

In hydrocephalus the pressure is usually high and the fluid is easily evacuated by lumbar puncture. Certain cases, however, those due to some obstruction to the normal outlets from the ventricles, may show only a low or normal pressure. The greatest help of a diagnostic nature is found in the study of pressure in cases of intracranial hemorrhage either from trauma or apoplexy. A bloody spinal fluid of high pressure is associated with concussion, fracture of the skull or other trauma, or with ventricular hemorrhage. This last may yield, however, a perfectly clear fluid. Capsular hemorrhage, on the other hand, exhibits, as a rule, a fluid water clear and of low or normal pressure.

Low Pressure.—Low spinal fluid pressure readings are quite frequent in cases with low blood-pressure, in collapse, and chronic debility. A low spinal fluid pressure, in cases exhibiting signs of intracranial pressure, denotes some obliteration of the upper part of the cerebro-spinal canal, as by exudate, impermeability of foramen of Magendie, or occlusion of foramen magnum by the brain itself. This situation is that which occurs in cases of hydrocephalus showing low pressure. It may also happen in acute serous effusions, œdema of the brain, as well as in the more chronic conditions, such as tumors of cerebellum or tumor of the posterior fossa. Often in these cases the first reading is high, but after the removal of one or more cubic centimetres, it falls very rapidly to below normal—showing no tendency to rise again. This is due to the entire blocking of the foramen magnum, which before was only slightly permeable. It is always serious, ending frequently with fatal result.

Serology has been developed primarily in the study of syphilis, and, therefore, in attempting to review the results in nervous and mental diseases it is well to divide the entire field into:

(1) Those conditions which are manifestations of syphilis.

(2) Those symptom-complexes which may or may not be an expression of syphilitic disease.

(3) The serological findings in those lesions due to organisms other than spirochæta pallida.

(4) Those conditions of unknown origin but which are never due to or associated with syphilis.

The first group includes all the clinical complexes which are directly traceable to luetic infection: syphilis, skin, visceral, nervous, tabes, and general paralysis. In infections an early recognition offers the best and often the only chance of successful therapy. The value of serology lies in the fact that by means of such examinations the disease may be detected in its incipience before any permanent tissue change has occurred.

At the time of infection, the period of the chancre, the only decisive diagnostic factor is the presence of the spirochæte in the sore. This is easily demonstrated in smears. The Wassermann Reaction of the blood remains negative, as a rule, until about the fourteenth day, when it becomes positive, increasing in intensity from then on until in the secondary stage it is strongly positive.

The spinal fluid has not been extensively studied during this period of the disease. The German literature has some records of spinal fluid examinations, but all with negative results. It is very probable that the invasion of the cerebro-spinal axis does occur very early in the disease in some individuals. Plaut reports a syphilitic cerebral meningitis in a patient whose initial sore had not healed; a spinal fluid Wassermann was not made in this case.

In the secondary stage, the stage of generalized infection, the serological findings are most decisive. Practically 100 per cent. of the cases give a strong positive Wassermann Reaction with the blood. The number of spinal fluid examinations made is still small, but of sufficient magnitude to prove without doubt that a small percentage show definite involvement of the nervous system. Ten per cent. of the cases studied show pleocytosis—that is,

more than ten cells per c.cm. Nonne in his studies thought that a moderate cell increase was probably present in 40 per cent. of all cases of infection, without denoting any involvement of the central nervous system. The globulin is increased, however, in about 13.3 per cent. of cases examined, and this always indicates the existence of organic changes in the brain or cord. The pressure is normal, as a rule, but may be increased; Wassermann Reaction of the spinal fluid is positive in between 10 and 15 per cent. of the cases. A number of definite cases of cerebro-spinal syphilis have been reported in men still presenting an eruption.

The majority of secondary cases examined in full give positive blood Wassermann; negative spinal fluid Wassermann; negative globulin test; positive Fehling's test; cells one to five per c.cm.; colloidal gold test is absolutely negative or inconstant. The significance of this gold reaction when present in the fluid is not known. Approximately 35 per cent. of the secondary cases examined showed some changes in the spinal fluid; about 10 per cent. have all the spinal fluid tests positive, the more usual combination of these latter cases being positive blood Wassermann; negative spinal fluid Wassermann; slight increase of globulin; negative colloidal gold; cells three to 35.

It is very important to note that in these cases showing definite abnormalities in the spinal fluid, practically none showed symptoms suggestive of involvement of the central nervous system. On the basis of the reported cases less than one per cent. of the patients with secondary syphilis show coincident cerebro-spinal symptoms, yet more than 35 per cent. show spinal fluid changes.

In tertiary syphilis the serology will vary definitely with the localization of the infections. Cases exhibiting skin or visceral lesions, as a rule, show normal spinal fluids—*i. e.*, with negative Wassermann Reactions; negative globulin; negative colloidal gold; positive Fehling's test; and cells one to five per cm. As soon, however, as the nervous system is involved, and usually long before there is any clinical manifestation of the involvement, the spinal fluid will become definitely pathologic, and the pathology of the fluid will vary with the anatomic localization of the luetic focus in the nervous system. Many serologists—Nonne, Dreyfus, Plaut, etc.—have attempted the establishment of formulæ for spinal fluid findings in nervous syphilis, fitted to these anatomic localizations. Syphilis may be predominatingly cerebral or spinal, or a combination of both. It may be acute or chronic. It may involve primarily and almost exclusively the meninges, inner walls of the arteries, or the brain and cord tissue itself. The serological findings in these different types of syphilitic lesions are more or less distinct, and while no hard and fast lines can be drawn as to the type of findings, the formulas occur with sufficient frequency to be of considerable diagnostic value. In acute syphilitic meningitis a turbid fluid may occur; more often it is clear. The pressure is almost invariably high. The cell count, made up for the most part of small mononuclear cells, is high, averaging between 150 and 3,000. The more acute the case the higher the number of polymorphonuclear cells found, and with the increase in polymorphonuclear cells comes the diminution in the strength of the Fehling's reduction test, which in some cases may be entirely negative. The Fehling's test is usually present, but where absent or diminished it always is indicative of a very active process. Globulin is

excessive and the spinal fluid Wassermann positive. The blood is also positive in about 90 per cent. of these cases. In the more chronic types of syphilitic meningitis the blood Wassermann is positive in about 90 per cent. of the cases; spinal fluid Wassermann is 60 per cent., using a routine of 0.2 c.c. of fluid for the test. With larger doses of spinal fluid 100 per cent. are found to be positive: the globulin test is positive and the number of cells varies from about 150 to 1,800. Fehling''s solution is promptly reduced.

When the syphilitic virus attacks primarily the inner lining of the blood-vessels—the endarteric type—the blood serum almost always gives a positive Wassermann Reaction. The spinal fluid gives a positive reaction in only about 60 per cent. Globulin increase is absent or slight: cells are normal border line or low pathologic count, 3–20. Fehling's reduction is always prompt.

In the gummatous form involving brain or cord tissue, the serology will vary according as there is more or less coincident meningitis. The average serology is positive Wassermann Reaction in blood and spinal fluids: cells varying from ten to one hundred; a positive globulin increase, that as a rule is not excessive; colloidal gold reaction showing the greatest precipitation of the gold in the dilutions one to eighty and one to one hundred sixty, and in these tubes it is very seldom complete. Fehling's reduction is prompt. The value of the colloidal gold test is in these cases considerable. It is frequently essential to differentiate between cerebral syphilis and paresis. In syphilis the spinal fluid Wassermann is much weaker, often not demonstrable with the smaller amounts of fluid. The paretic spinal fluid Wassermann is usually very strongly positive with 0.2 c.c. of fluid. The cerebral syphilis gold curve is, as a rule, 0.013.310.000, while the paretic curve is almost invariably 5555543.100. There occur cases, clinically identical with paresis, in which all the serological examinations are those of cerebral syphilis, and again cases which clinically would be classed as cerebro-spinal syphilis whose blood and spinal fluid are of paretic type. There are not sufficient autopsy records to decide decisively on the relative merits of these reactions in cases of this kind.

1. In tabes dorsalis the blood and spinal fluid examinations are of great importance, especially in the pre-tabetic stage—*i.e.*, when the patient has not well-defined neurological symptoms, but complains of vague pains—rheumatism, stomach and intestinal troubles, etc. These symptoms should always call for serological examination, especially if in the background is a syphilitic history. The serological findings in these cases are of more diagnostic importance than the Argyll-Robertson pupils and absent knee jerks, because they are demonstrable sooner. In a clear-cut, full-fledged case the laboratory is not nearly so valuable. The more common findings in tabes dorsalis is Wassermann positive in blood; negative in spinal fluid, except where large doses of spinal fluid, 0.5 to 1 c.c., are tested. The globulin reaction is negative or only weakly positive; cells varying from 25 to 100; Fehling's reduction is prompt; colloidal gold giving inconstant results, entirely negative or the luetic type of curve. Many cases, however, may have negative blood; positive spinal fluid; while still others may have all

reactions and tests positive; the cells—50 to 80 per c.cm., and a colloidal gold curve which may be leutic in type or show a distinctly paretic curve. Whether this last should really be classed as tabo-paresis cannot be cleared without further autopsy studies; certainly many cases with this form of serology show no symptoms whatever of paresis.

In paresis, again, the period of extreme importance for serological examinations is long before the symptoms of paresis are established. All careful serologists and clinicians to-day are convinced that little is gained in the treatment of fully developed paresis, either clinically or serologically.

All vague symptoms occurring in an individual known to have had a luetic infection should excite suspicion and call for full serological examination. In the longer or shorter pre-paretic stage much could be done if the conditions were discovered. The incipient stage of paresis has no fixed formula. No one grouping of blood and spinal fluid findings means absolutely beginning paresis. The more usual result of these examinations based on a large series of cases that have ended as typical cases of general paralysis, was a strongly positive Wassermann Reaction of the blood, that was influenced very little, if at all, by intensive treatment—*i.e.*, a persistent strongly positive reaction in blood; a negative reaction in the spinal fluid, associated with moderate increase in the globulin content; a cell increase, as a rule, of more than 60 cells; a colloidal gold test showing complete percipitation of the gold in the dilution 1 to 10, 1 to 20, 1 to 40, 1 to 80, and 1 to 160.

In a full-fledged typical case of general paralysis every reaction is positive. The Wassermann Reaction in both blood and spinal fluid is very strongly positive, comparable in intensity only with the reaction as it is found in secondary syphilis at the height. The lesions here are parenchymal. The spirochætes are placed deeply in the brain substance. The meningeal irritation is slight and the globulin increase and cell count (17 to 50) moderate. Fehling's solution is promptly reduced; the cells found in the fluid are practically 100 per cent. lymphocytes. The colloidal gold test is very decisive; the average being complete precipitation charted empirically as 5, in the dilution 1 to 10, 1 to 20, 1 to 40, 1 to 80, 1 to 160, and 1 to 320; moderate precipitation 1 to 640; less in 1 to 1,280. None in other tubes.

In the late stages of paresis, the stage of complete dementia and general decline, the reactions may all continue positive or they may all become negative. They may become entirely negative with treatment—slight, moderate or intensive—or with no treatment whatever.

Mitchell, Newcomb and Darling reported a series of spinal fluid, cell counts, etc., made in untreated paretics. The fluid was taken every two weeks for a period of some twenty weeks. The highest counts occurred in full-fledged cases, especially those showing excitement and grandiose tendencies; but they also occurred in some patients with marked dementia and in some with comparative remissions. In some of their individual cases very great variations in the counts occurred from week to week, as 30 to 150; 2 to 15; 0 to 30; 0 to 60—with apparenty no change in the patient's condition. It is evident from their reports that a reduction to a normal count may occur in this disease without any treatment, other than the drainage.

Many reports of cure in paresis have appeared in the literature based

on the absence of the ordinary reactions, the dementia being explained as the result of the previous disease. In judging the result of any treatment it is essential to remember the tendency to remission in this disease with and without improvement in the serology, and to variations in the serological findings with and without any clinical improvement.

A low count may arise and continue for months. The globulin excess also may vary. The most common serological combination, however, in the late stages of paresis is negative blood Wassermann Reaction; positive spinal fluid Wassermann; negative globulin excess (or, if present, a weak reaction); cells, 35 or less—often none; a prompt reduction of Fehling's solution, and a colloidal gold reaction which retains its typical paretic type.

Definite conclusion concerning treatment should, therefore, not be based on fall in cell count, globulin or even Wassermann reactions, unless there is a coincident and permanent clinical improvement, especially when it is realized that falling counts and loss of other reactions are not uncommon before death in paresis and occur very frequently in rapidly progressive cases.

About 3 per cent. of the patients examined may be entirely negative in both blood and spinal fluid before death and show spirochæte in the brain tissue at autopsy.

In congenital syphilis the serology is the same as in the adult type. Under treatment the tests of blood and spinal fluid tend to become normal. The closer the treatment to the initial infection the more prompt and permanent is the result. With the intravenous injection of Salvarsan, the blood Wassermann generally disappears first. With the Swift-Ellis intraspinal treatment the cell count is first influenced, probably as much by the removal of the fluid as by the administration of the serum. The result of the treatment, as evidenced by the return of blood and spinal fluid to normal findings, is least encouraging in tabes and general paralysis. All exudative conditions are, as a rule, promptly influenced by therapy—both serologically and clinically, although there is no hard and fast rule as to which finding will disappear first.

2. The next great value of serological examination lies in those conditions which may or may not be caused by syphilis. When syphilis is the etiological factor the examinations will give the ordinary leutic results, previously described. In a few conditions, other tests have been devised which add some decisiveness to the results, but as a rule the only rôle of serology is to eliminate syphilis as a cause.

An endarteric syphilis may give rise to hemiplegia—embolism, thrombosis, cerebral softening, or to the cerebral palsies of children with the typical serology of spinal syphilis. When due to any other cause the results of the blood and fluid examinations in all these lesions are uniformly negative.

Hydrocephalus usually is entirely negative. In acute cases there may be a marked cell increase and the globulin may be excessive. Pressure is always high. In post-traumatic hydrocephalus there is an increased pressure and increased amount of fluid, but cells and globulin are generally normal. In concussion with fracture, however, there may be a clear or bloody fluid, increased pressure, and cells that may or may not be increased; globulin is

usually normal, but may also be increased. It will always be excessive in bloody fluids.

In fracture with infection the fluid becomes turbid; pressure very high; globulin markedly increased; bacteriological examination shows the infecting organism.

A gumma or tuberculoma may give rise to the clinical picture of brain tumor, and the gumma gives the ordinary leutic serology. A true brain tumor is usually associated with a normal blood and spinal fluid—the latter under increased pressure. Rarely there may be a slight increase in cells, seldom above the border line counts. A globulin increase may be present or absent, dependent upon the position of the tumor and the presence or absence of meningeal irritation.

The diseases of the spinal cord—excluding cerebro-spinal syphilis, tabes dorsalis and inflammations—are practically all entirely negative serologically.

Combined sclerosis gives entirely normal results with the routine serological examination, but is usually associated with more or less severe blood changes. A few cases show an increase in the globulin content of the spinal fluid unassociated with a cell increase. This type of spinal fluid may be found also in syringomyelia, hematomyelia, disease of the caudæquina, tumor, Pott's disease, and a few other conditions.

The serology of spinal cord tumors will depend on the nature of the tumor, a gumma giving the ordinary luetic reactions. The compression syndrome of Nonne is a not uncommon finding—that is, the lemon yellow color xanthochroma, enormously increased globulin reaction with little or no increase in cells. Most of these fluids coagulate spontaneously. Fehling's reagent is at times not reduced.

Involvements of the nerves are almost invariably negative to luetic tests. Spinal fluid findings are entirely negative, herpes zoster alone at times showing a moderate cell increase—3 to 20 cells.

Functional neurosis and insanities may occur in syphilitic persons, but in those not leutic the serology is uniformly negative. This is true also of epilepsy, tetany, chorea, paralysis agitans, Graves's disease, myxœdema, acromegaly, myasthenia gravis, etc.

Aside from those conditions, which now and again are caused by or associated with syphilis, there is a large group due entirely to infections which have a serology of their own.

3. In the infections of the meninges the Wassermann Reaction is in all save syphilitic meningitis negative in both blood and cerebro-spinal fluid. The globulin is always increased, and increased as a rule to a greater extent than is found in any case of syphilis. The cell count ranges from 100 to 30,000, often too many for accurate count without dilution. The highest cell counts observed occur in infections due to the streptococcus. The cells, except in tubercular meningitis are predominantly polynuclear; Fehling's solution is usually not reduced; colloidal gold tests are very irregular and of little value. Most meningitic spinal fluids precipitate the gold in the tubes of higher dilution, as 1 to 320 and 1 to 640.

In these cases the most important part of the examination is the study

of the cytology and bacteriology. In the majority of cases the bacteria can be demonstrated in smear preparations, and yet in all cases cultures should be made. In tubercular meningitis the fluid may be clear or faintly opalescent; globulin is increased and the increase can be demonstrated even after diluting the fluid five times. The cells are small mononuclear lymphocytes and may vary from 60 to 30,000, usually between 200 and 300. Organisms may be found in smears; more often none are found or found with great difficulty, and animal inoculations will be necessary. In meningococcus infection—epidemic cerebro-spinal meningitis—the cells for the most part (polynuclear) rise to several thousand per c.cm., often too abundant to accurately count. The globulin is greatly in excess. Other proteins are also increased to demonstrable amounts. Fehling's reduction is absent. Smears made from the sediment show numerous gramme negative intracellular diplococci. These grow on Löffler's blood serum or ascitic broth containing 1 per cent. dextrose. Blood serum of infected cases contains specific agglutinins for meningococcus. With pneumococcus infection, a full-fledged meningitis is common with typical meningeal fluid reactions and yielding pneumococcus on culture. It is possible to have during a general infection a spinal fluid from which pneumococcus can be cultured, but without any clinical signs of meningitis. The spinal fluid may or may not have a cell increase or globulin increase. It may be turbid or clear. The great majority of cases of pneumonia, however, have sterile spinal fluid.

Besides the meningitis due to meningococcus, pneumococcus and tubercle bacilli, exactly similar conditions with the same kind of spinal fluid findings occur with infection by bacillus of influenza, diphtheria, typhoid, paratyphoid, staphylococcus, streptococcus, and more rarely B. coli, B. pyocyaneus, B. mallei, B. anthracis, saccharomyces, actinomyces. The globulin and cell content are uniformly increased, the degree of excess varying with the virulence of the invading organism. Meningitis has also been caused by trichina spiralis.

This type of spinal fluid also occurs to a lesser degree in cerebral abscess. If the abscess is well encapsulated the fluid may be entirely negative. The degree of spinal fluid changes, as a rule, is dependent on the amount of meningitic reaction. A negative result can also occur when communication with subarachnoid space is shut off. Cell count may then be border line and globulin excess very mild. Fehling's reduction test is positive, except in fluids which become filled with pus cells.

Encephalitis and cerebritis have followed mumps, erysipelas, typhus, malaria, influenza, and pneumonia. It is not uncommon after infected head wounds. Symptoms are those of meningitis. There is an increase in cerebro-spinal fluid; an increased spinal pressure; the cell count of the spinal fluid is never very high; globulin increased but never excessive; cultures of blood and cerebro-spinal fluid may be, and generally are, sterile.

In myelitis sterile fluids are more often found than those yielding organisms on culture. Cells may be normal or very slightly raised; globulin is generally very little increased; the blood cells usually show changes; blood cultures are usually negative; Fehling's reduction test is weak or absent.

In anterior poliomyelitis cultures on ordinary media are negative. The

fluid is infective because of a filterable virus which it contains. Wassermann Reaction in blood and spinal fluid is always negative. At an early stage of the disease, especially before the onset of the paralysis, the spinal fluid shows a moderately increased cell count with a low or normal globulin content. Polynuclear cells may represent at this time at least 90 per cent. of the total, but usually the cells are almost entirely made up of lymphocytes and large mononuclear cells. After the first two weeks the cell count drops to normal or nearly normal and there is frequently an increase in the globulin which persists for seven weeks or longer. All fluids reduce Fehling's solution.

There exists one other group of meningeal conditions where the serology is more or less distinctive. These are hypertrophic spinal meningitis and pachymeningitis hemorrhagica. The Wassermann Reaction in the blood and spinal fluid is always negative. There are no cells, but a marked excess of protein, albumen and globulin, which coagulates on heating. In pachymeningitis hemorrhagica the fluid may have a distinct pink color, due to blood coloring matter.

4. In all the other neurological or psychiatric conditions serology offers little of moment. Many studies, as H-ion content, urea, uric acid, cholesterin content, etc., have added to our sum total of knowledge but yielded nothing of clinical help. From these clinical studies a few facts have been collected. In uræmia there may be an increase in chlorides in the cerebrospinal fluid without a corresponding increase in urea, while in other cases there may be an increase in urea. In diabetes the glucose is often markedly increased. Acetone is frequently present and diacetic acid has been found, but only in very profound intoxications. Acetone has also been found in cases of Addison's disease.

Résumé.—Serology which includes examination of blood and spinal fluid establishes:

1. By means of the Wassermann Reaction:
   (a) A method of diagnosing syphilis;
   (b) Of eliminating syphilis as a causative agent;
   (c) Of detecting syphilis as a complicating factor.
2. By means of the colloidal gold reaction:
   (a) Method of differentiating paresis from less malignant forms of syphilis.
3. By means of the globulin test and cell count:
   (a) A method of differentiating organic from functional diseases.
4. By means of bacteriological examination:
   (a) Determining the causative agents in cases of infections.

Allergy: Anaphylaxis: Serum Sickness.

These terms are used in a general way to designate the train of symptoms caused by protein poisoning.

Foreign proteins, though constantly introduced into the alimentary canal, do not under normal conditions gain access to the fluids and tissues of the body. To do this they must first be acted upon by the proteolytic ferments of the digestive fluids and split up into less complex bodies as peptons

and amido acids, in which form they pass through the mucosa of the digestive tract to gain access to the interior of the organism.

Under certain circumstances, however, foreign proteins reach the fluids and tissues of the body without having undergone changes in their constituent form. This, as Vaughan has pointed out, occurs in three different ways:

First, As the result of abnormal permeability of the intestinal mucosa in certain individuals for particular proteins. Under this heading are to be grouped the various food idiosyncrasies described further on. The striking peculiarities of this form of protein poisoning are the rapidity with which the symptoms develop after the ingestion of the offending article of food, the minuteness of the quantity capable of giving rise to intense symptoms, and the specific nature of the poisoning as manifest in the prominence of cutaneous and respiratory manifestations.

Second, Parenterally, that is to say, not by way of the mucosa of the digestive tract, but directly into the fluids and tissues by experimental injection or the use of therapeutic sera.

If a normal guinea-pig receives a small amount of normal horse serum by injection subcutaneously, intravenously or into the peritoneum no apparent change takes place. Nevertheless remarkable effects have been produced in the fluids and tissues which do not manifest themselves until a later period and under definite specific conditions. If, after several weeks, the animal thus treated is again injected with the same dose of normal horse serum, urgent symptoms immediately occur and death may follow. The horse serum, which upon the first injection had no manifest effect, has become on the second injection an intense poison. A proper interval of time must elapse between the two injections for the manifestation of these changes. If the re-injection is performed in the course of a few days no toxic phenomena result. This " condition of unusual or exaggerated susceptibility of the organism to foreign proteins " (Rosenau) has been designated anaphylaxis.

Third, Foreign protein may enter the body as the result of bacterial infection. Under these circumstances the cellular elements which constitute the proteins have the capacity of growth and multiplication after their introduction into the body. During this process certain body-cells are stimulated to the production of an enzyme capable of destroying the pathogenic organism. "As the bacterial cell is broken up under the influence of the special enzyme, poisonous products are liberated which exert their harmful action and give rise to the symptoms of illness. This special ferment, once formed, remains in the body after recovery from the disease, and is stored up in certain cells as a zymogen for future use. When subsequently a bacterial cell of the same species again enters the body, its presence at once reactivates the zymogen already formed, and consequently the invading organism is destroyed before it has had opportunity to grow and multiply to any extent. As a result of this fact the individual once affected has acquired an active immunity to subsequent attacks of the same disease." [1]

Allergy.— Von Pirquet, basing his conclusions on a series of clinical observations, suggested this term, which literally means altered reaction, to designate the phenomena under consideration without indicating any theo-

[1] Vaughan, V. C., Jr. Internat. Clinics, vol. IV, 21st series, p. 142, J. B. Lippincott Co., 1911.

retical explanation for them. In view of the complexity of the subject, the enormous amount of research carried out, and the abundance of new facts collected, without the attainment up to the present time of any satisfactory conclusive generalizations, it would have been better to have retained the word "allergy" in preference to anaphylaxis. Allergy has been employed in a different sense to indicate the effort of the body to localize the activity of the spirochæte, which tends to be generally distributed in syphilis, after prolonged infection; an effort sometimes seen in the early secondary period, but more usual in the tertiary stage, when the lesions are not infrequently limited to one or two areas.

Anaphylaxis.—This term, meaning "without protection," was introduced by Richet, and has come into general use, though at the present time its appropriateness may well be questioned, since the actual condition is regarded as in many cases favorable to the development of immunity. It is differently employed by writers upon the subject. Some use it to designate the anatomical and functional changes which occur when an animal receives under the required conditions a second injection with the same protein solution. Others employ it to describe the sensitized state produced by the first injection of some foreign soluble protein, while others again apply it to the sensitization produced by the first injection and the intoxication caused by the second injection of the same protein.

The term is not applicable to the toxic reactions which occur when any one of a large number of very different substances are injected; nor should it be used to designate the condition when the injection is repeated in three or four days—that is, before the expiration of the definite period—and the animal remains perfectly normal. It is the development of characteristic toxic phenomena upon the second injection of the same soluble protein after the expiration of the appropriate interval that constitutes anaphylaxis.

This being the case, anaphylaxis may be considered in three different phases: (*a*) sensitization; (*b*) incubation; and (*c*) intoxication.

(*a*) Sensitization may be caused by any soluble foreign protein, either animal or vegetable, introduced in an unaltered state into the circulatory fluids of an animal. The list is a very large one and comprises: 1, animal proteins in solution; 2, cellular animal proteins; 3, vegetable proteins in solution; 4, cellular vegetable proteins. The sensitizing dose varies in different animals. The most susceptible animal is the guinea-pig, but it is probable that every species of animal may be sensitized. Other ordinary laboratory animals, as the rabbit, are less susceptible. In the case of the guinea-pig the dose of horse serum or crystallized egg albumen is practically infinitesimal. The most certain method of inducing laboratory sensitization is the injection of the selected protein subcutaneously, intravenously or into the peritoneal sac. Other methods consist in feeding the proteid substances to animals, their inunction into the scarified skin, and their repeated introduction, particularly that of horse serum, into the nares or into the vagina or rectum of guinea-pigs. It has also been established that sensitization in the case of guinea-pigs may be transmitted from the mother to her offspring. These experimental proofs that sensitization may be inherited or acquired by the contact of proteins with the uninjured

mucous membranes or integuments may be invoked to explain the more or less violent anaphylactic reaction which sometimes occurs in human beings upon the first injection of an antitoxin for therapeutic or immunizing purposes and certain idiosyncrasies.

That sensitization is specific is fully established by the fact that a reaction can only be obtained by the reinjection of the same protein used in the first injection or a protein derived from an animal or vegetable of a closely related group.

(*b*) The incubation is the interval required by the body fluids and tissues to undergo these changes which endow the soluble foreign protein, practically harmless upon the first injection, with the properties of a virulent poison. Sensitization develops gradually and reaches its maximum at varying periods in different animals. In the guinea-pig the incubation is complete in about ten days; in the rabbit, eight to fifteen days after the last of several injections (Arthus phenomenon); in man, seven to twelve days. The duration also varies with different animals. In man typical reactions have occurred upon reinjection after five years.

(*c*) Intoxication. It is in this phase that anaphylaxis declares itself. The period of incubation may be completed, and sensitization gradually and fully established, to gradually subside after a shorter or longer interval; but the animal remains practically normal unless it is injected with the same protein. When this happens acute or subacute symptoms of intoxication promptly supervene and the requirements of the concept designated anaphylaxis are fulfilled. These symptoms vary in different animals, but are practically constant in the same species, whatever the protein may be that causes them. In man cutaneous lesions are common and usually accompanied by respiratory and circulatory derangements.

In the laboratory intoxication is brought about by the same methods by which sensitization is caused: in man by the inhalation or ingestion of the sensitizing protein or by the use of immunizing sera. In general terms, the quantity of foreign protein required to cause intoxication is much greater than the quantity which sensitizes.

It has been generally accepted that the protein molecule acts both as the sensitizing and the intoxicating substance. Vaughan has shown that proteins are split into a toxic and nontoxic fraction by prolonged exposure to a high temperature in a 2 per cent. solution of sodium hydrate in absolute alcohol. The toxic fraction causes fatal anaphylactic phenomena in guinea-pigs but does not sensitize them. The non-toxic fraction causes sensitization which reacts to the entire protein molecule but fails to react to the non-toxic fraction. This experimental work forms the basis of the parenteral digestion theory of anaphylaxis formulated by Vaughan and is regarded as the most acceptable explanation of the subject presented up to the present time. Its main points have been set forth in foregoing paragraphs.

Anti-anaphylaxis according to this view constitutes a condition in which there is a disproportion between the amount of the specific ferment and the foreign protein, since the anaphylactic reaction calls for much of the ferment, and the remainder is incapable of releasing a sufficient quantity of the poison to cause reaction. Passive anaphylaxis is the condition

caused by the transference of the specific proteolytic zymogen from a sensitized animal to a normal one.

The intoxication symptoms in the human being comprise cutaneous eruptions, severe arthalgias, swelling of the lymph-nodes, fever of remittent type, œdema, albuminuria and leucopenia.

Serum Sickness.—This term was applied by von Pirquet and Schick to the well-defined symptom-complex of anaphylactic reactions which sometimes occur in man after the injection of therapeutic sera, most of which are obtained from the horse.

These symptoms occasionally occur not only after injections of antidiphtheritic, antistreptococcic and other sera, but they may also follow the injection of normal horse serum. They are therefore not due to the antibodies present in the serum but to the serum itself. It has also been established that these reactions are more common in cases treated with large amounts of serum than with small amounts of serum containing the same number of antitoxin units. It has further been shown that serum derived from certain horses is more frequently followed by anaphylactic reactions than of other horses injected in similar amounts.

In a majority of the cases the typical intoxication reaction does not occur until the end of an incubation period of about eight to twelve days. Among the earlier symptoms are fever, which may last from a few days to a week or two; enlargement and tenderness of the lymph-nodes in the area of the injection; and eruptions at first urticarious, subsequently of the most polymorphous description and symmetrically distributed. During the incubation period there is a moderate leucocytosis, which gives place to a marked but transient leucopœnia. Joint affections are less common and involve usually the metacarpophalangeal articulations, the wrists and the knees. They are painful, but tend to recovery without disability or deformity. The œdema appears first in the face; later in the dependent parts of the body. There is also a slight albuminuria. The subsidence of the glandular swelling and disappearance of the œdema are of favorable prognostic significance, since they are shortly followed by recovery.

In an individual who has had previous injections of horse serum this typical reaction is modified in respect of the time of its occurrence. There are two types of modification: First, an immediate reaction, which occurs within twenty-four hours, and second, an accelerated reaction, which occurs about the fifth or seventh day. When the injection is made in from ten days to six months following the primary injection the immediate reaction commonly appears; after six months the accelerated reaction. In other respects the reactions are similar to those of a primary injection, but run a shorter course.

As a rule the serum sickness in man, as characterized by the foregoing symptoms and manifesting the immediate and accelerated reactions, runs a favorable course and terminates in recovery. It has occasionally occurred, however, that a violent or fatal anaphylactic shock has promptly followed the first injection of horse serum, even in small amounts. These accidents are fortunately of infrequent occurrence. The symptoms resemble those of anaphylactic shock in laboratory animals, and it may be assumed that they are due to similar causes. The larger proportion of the cases have

suffered from intense dyspnœa and convulsions, while the pulse continued for a time full and regular. At autopsy in some instances the lungs were found overdistended. These manifestations closely resemble the fatal anaphylactic shock of the guinea-pig. A history of asthma or other respiratory affections usually has been obtained. A careful anamnesis bearing upon these conditions, and especially upon the occurrence of asthmatic symptoms following exposure to the effluvia from horses, should be made in cases in which injections of sera are indicated. In a more limited group of cases the first injection of horse serum has caused anxiety, profound depression, cyanosis and great feebleness of the action of the heart-collapse symptoms such as occur in the anaphylactic shock of the rabbit and dog. Many of these patients have never received a previous injection of horse serum. The intense symptoms and acute death cannot be attributed to an essential toxicity of the serum, for first injections of horse serum not only as a general rule do not cause anaphylactic phenomena, but they usually do not produce immediate symptoms of any kind. The reactions are distinctly those of an anaphylactic intoxication of the most violent character and develop with characteristic promptness. It must be assumed that they are due to a sensitized condition of the subject, the result of the parenteral digestion of unchanged horse protein absorbed by the intestinal tract or in one of the other ways by which laboratory animals undergo experimental sensitization.

The recurrence of hay fever, hay asthma, horse asthma and the various food idiosyncrasies are instances of anaphylaxis of the milder type. There is reason to suspect that all cases of asthma are manifestations of protein sensitization, and that the common occurrence of asthmatic seizures in persons suffering from chronic affections of the respiratory tract results from an anaphylactic reaction to the proteins of the infecting organisms by which such affections are caused.

Idiosyncrasy.—This term has long been used to describe the condition in which the inhalation of the effluvium of animals, especially the horse, rabbit or guinea-pig, or of the pollen of a great variety of different plants, or the ingestion of certain articles of food, among which may be particularly named eggs, cheese, pork, shell-fish, buckwheat, and strawberries, is directly followed by urgent and distressing symptoms. Asthma and urticarial rashes are almost constant, but abdominal distress and pain, vomiting, diarrhœa and prostration are common and sometimes severe. This condition of hypersensitiveness is anaphylactic in character and specific, antibodies for the particular protein being present in the cells and fluids of the affected individual. It is also extreme, as the most intense symptoms often follow the inhalation of exceedingly minute amounts of protein in horse-asthma, while the instillation of a few drops of a 1 per cent. physiologic salt solution of pollen into the lachrymal sac of a susceptible individual is sufficient to cause an attack of hay-fever. This form of anaphylaxis is common and often hereditary. The circumstances under which sensitization has taken place are mostly unknown. The sufferer is usually aware of the particular animal, pollen or article of food which causes his attacks and is on the watch to avoid it.

Some persons are sensitized to particular drugs, among which are

atropine, strychnine, morphine, iodoform, ipecac and certain preparations of iron, and suffer severely after exceedingly small doses. If under these circumstances the drug in question does not contain a protein substance it is necessary to assume that it may act upon a body protein and convert it into a foreign protein, capable of sensitizing other cells of the body.

The diagnosis of idiosyncrasy rests upon the fact that the patient almost invariably upon exposure to the emanations from particular animals, the inhalation of certain pollens or the ingestion of definite articles of food or drugs suffers at once from the characteristic train of symptoms.

Cutaneous and intracutaneous tests are successfully employed in the case of food idiosyncrasy. The technic of the cutaneous tests consists in cleansing the skin of the arm with alcohol, abrading a small area by a needle or other suitable instrument and rubbing into the abraded surface a minute portion of the suspected article, as a drop of egg albumen or a drop of a 5 per cent. watery solution of the food. A positive reaction appears in ten minutes or less in the form of an urticarial wheel surrounded by an erythematous areola, and lasts from twenty to forty-five minutes. A control must be made with water or salt solution, since the trauma itself may be followed by some œdema and redness in persons with an irritable skin.

The intracutaneous test consists in the injection into the skin of a minute amount of a sterile solution of the protein of the suspected food—0.1 c.c. of 0.1 per cent. solution is usually sufficient. A positive reaction is seen in a tender papule, with œdema and an erythematous areola. This test is very delicate but somewhat painful. Confusing non-specific reactions are common, but subside in a short time. For this reason the readings should not be taken until after forty-eight hours. Soluble proteins of foods are prepared for these tests in the biological laboratories and sold in the shops. The offending food may not always be found at once and a series of tests may then become necessary.

The prognosis may be favorable if the offending protein is found and desensitization undertaken by the systematic feeding of the offending food in minute and non-toxic doses with each meal until the skin reaction becomes negative; the same result may be attained by the subcutaneous injections of sterile solutions of the offending protein, beginning with minimal doses and increasing by degrees.

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